DOES NOT CIRCULATE

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Editorial

Fluorescent Microscopy— A New Aid in Diagnostic Laboratory Medicine

Although the technique of fluorescent microscopy is not new, recent laboratory applications of the method have revived it as one of the most exciting and promising techniques in medicine today. New uses are described in practically every journal including this issue of *The Journal** and for this reason a brief description of the method with mention of present day tests and future uses might be helpful to the busy clinician.

Fluorescent microscopy is based on the principle of "excitation" of certain fluorescent chemicals by exposing them to light rays of a particular wave length. The term "fluorescent" means that the substance has the ability to emit light rays after exposure to other light rays. The emitted rays are visible and they are longer than the absorbed rays. Hence, if we direct invisible (ultra-violet) rays through tissue, smears, or other specimens which have been exposed to a fluorescent dye, any particle on the slide that becomes stained or combines with the dye will "fluoresce" or emit visible light. Therefore, it will stand out as a focus of light in a dark background. Different fluorescent dyes for specific substances are being reported. When a slide is covered with biologic material and is exposed to the dye, only the specific substance will combine with the dye and become fluorescent. The excess of flourescent dye is then washed off the slide. The slide is examined under the microscope with an ultra-violet light source ("flourescent microscope"). Foci of fluroescence appearing on the slide would confirm the presence of the specific substance in question. Absence of fluorescence would indicate that all the dye was removed in the wash and therefore, no specific substance was present.

What are the applications? These are almost innumerable and certainly very interesting. The huge field of antigen-antibody reactions offers us excellent possibilities, not only for research, but practical laboratory applications. Our present laboratory methods in allergy, hypersensitivity, and immunology are fairly crude when compared with the methods of fluorescent microscopy. In the present methods we must depend on combination of the antigens and the antibodies in a test tube in sufficient quantities to form a particle that will either precipitate, agglutinate, or combine with complement. (These are the best present-day methods.) Almost ideal conditions must be established for these methods to work in certain diseases. In virus diseases the antigens are too small to be seen. In certain tests, the antigen used is not even composed of the infectious agent, but is an extract of some other tissue which has been demonstrated to react with the antibody. (Serologic test of syphilis.) These tests vary in sensitivity and specificity. The complement fixation test is probably the best of this group; and it is time consuming and expensive. The fluorescent antibody technique, when properly controlled, has the inherent specificity of an immuno-chemical reaction. In essence, the method is as follows:

1) Specific antibody is developed by immunization of a suitable laboratory animal.
2) The specific antibody is extracted and concentrated by recognized chemical methods.
3) The fluorescent dye (usually fluorescein) is chemically combined with the antibody.
4) The biologic material which is suspected of containing the antigen in question is placed on a slide and is then exposed to the fluorescent antibody solution. The slide is washed to remove excess fluorescent antibody. Only antibody that is combined with antigen remains on the slide. The slide is then examined under the fluorescent microscope for the presence or absence of fluorescence and hence, the presence or absence of antigen is demonstrated.

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An actual example demonstrates the relative simplicity. Assume we have a patient with questionable influenza. (Influenza is used as an example because virus diseases, in general, are most difficult to diagnose specifically with our present day methods, and it is anticipated that the fluorescent microscopic techniques will adapt themselves admirably to the diagnosis of virus diseases.) A smear is made of the secretions of the nasopharynx (the localization of the virus). The smear is flooded with solutions containing fluorescent anti-influenza antibody. The smear is washed and examined; fluorescence confirms the diagnosis and the absence of fluorescence disproves it. The test is completed in two hours or less. If an attempt were made to culture the virus, or if efforts were made to demonstrate an antibody titer, several weeks would be required for diagnosis. Up to the present time, approximately 20 different viral antigens have been localized in infected cells. The method also lends itself to nearly every other form of antigen and antibody identification, including various hypersensitivity antibodies in tissue, and identification of fungi, bacteria, and Rickettsia.

Fluorescent microscopy is also finding use in the diagnosis of cancer. The following principles are utilized:

1) The two types of nucleic acid are present in the cells, (a) deoxyribose nucleic acid. (DNA), which is the characteristic chemical component of the chromatin of the nucleus and (b) ribose nucleic acid (RNA), localized mainly in the cytoplasm and nucleolus.
2) A high amount of RNA is generally characteristic of the cells showing high protein synthesis, including malignant cells.
3) The fluorescent dye, acridine reddish orange (AO), causes green fluoresence with DNA and red flourescence with RNA. Hence, the malignant cells are distinguished by their red fluorescence. This method has been utilized in screening cervical smears for cancer cells. It has also been used for staining tissue.

Many technical problems must be solved before fluorescent microscopy can be used routinely in the laboratory. At the present time, fluorescent antibodies are not available commercially. Each laboratory must prepare its own antibody solutions. This is a difficult and time consuming procedure, and few laboratories are capable of doing it well. The microscopic apparatus is expensive and few laboratories own it. Ultraviolet light is dangerous to the skin and eyes, and personnel must be trained in the correct use of the fluorescent microscope. However, as is true of all valuable technical procedures, these and other problems will be solved and the cost will be reduced. When this occurs numerous new developments and extension of present research techniques may be expected in fluorescent microscopy. These might include some of the following:

1) Rapid and inexpensive screening of large numbers of cytologic smears with reduction in the cost to the patient.
2) Application of the cytologic method to areas of cancer diagnosis where it is not used due to the degeneration of cancer cells or inability of the cytologist to identify the malignant cells because of their minimal changes (GI tract, prostatic secretions, urine sediment.)
3) The pathologist may utilize the technique in trying to specifically diagnose malignant cells of an unusual nature ("lateral aberrant thyroid," bronchial "adenoma," etc.) by chemical changes when the histological changes are equivical.
4) Identification of specific infectious agents will be done rapidly and accurately in epidemics as well as on routine sporatic cases.
5) The method will be used for ruling out or specifically identifying infectious agents in tissue.
6) It will be used for the localization of antibody production in cells.
7) It can be used for localization of specific protein substances in the body.
8) The technique may be used to prove the presence of antigen-antibody reaction in certain diseases and demonstrate its specific localization. This will be helpful in establishing the etiology of certain diseases that have been suspected of being allergic in origin, but which have not been proven to be by our present methods.—*HTR.*

Contraindications for Radical Mastectomy

Conventional radical mastectomy is the procedure of choice for treatment of carcinoma of the breast. The malignancy is curable in a significant percentage of cases, and the operation is attended with low morbidity and mortality. In the presence of evidence that the disease has spread beyond the confines of surgical removal, Roentgen therapy and/or hormonal therapy should be employed to achieve maximum palliation.

Considerable difficulty lies in accurate evaluation of patients in whom cancer of the breast is present without evidence of distant metastasis but in whom there are local signs of advanced disease. If these signs are ignored and radical operation performed, the operation will fail to alleviate the symptom of the patient and probably hastens her demise.

The criteria of Haagensen and Stout[1] for categorical inoperability were based upon a classical study of results of the treatment of carcinoma of the breast at Presbyterian Hospital, New York, in the years 1915 to 1934. These were revised slightly after a similar study in the same institution in the years 1935 to 1942. Stated briefly, they are:

(1) Extensive edema over the breast
(2) Edema of the arm
(3) Satellite nodules in the skin over the breast
(4) Intercostal or parasternal nodules
(5) Proved supraclavicular metastasis
(6) Inflammatory carcinoma of the breast
(7) Distant metastasis to the lung, skeleton, etc.
(8) Two or more of the following:
    (a) Ulceration of the skin
    (b) Local edema, less than one-third of the breast surface
    (c) Fixation of the tumor to the chest wall
    (d) Large axillary nodes with fixation

These criteria have stood the test of time and their usefulness has been proved most strikingly by those who choose to ignore them.

It is noteworthy that of age, neither youthfulness nor senility, contraindicates a surgical attempt for a cure. It is encouraging that pregnancy and lactation have been dropped from the list of contraindications though certainly the confluence of these circumstances lessens the probability of survival. The engorged breast of gestation and lactation make correct evaluation of a tumor mass difficult both for the patient and her attending physician and probably accounts for the delay in diagnosis and treatment.

Biological predeterminism is an interesting concept which implies that pathologically identical carcinomas of the breast may behave in a strikingly different manner. One malignancy will pursue a fulminating lethal course not favorably altered by operation at any time from its inception. Other breast cancers are more kindly in that their host relationship is such that they may be cured even late in their life history by simple surgical means. Another group of breast cancers comprises the target for which classical radical breast surgery has been aimed in the hope that early diagnosis of the lesion and adequate surgical removal may truly increase their curability.

Stated differently, this disillusioning and disturbing theory would show that all small breast cancers are not early and curable and that all large breast cancers are not late and incurable.

The validity of this theory is not in question but believe whole heartedly that its proof is best expressed in the difference in philosophy of individual surgeons. Depending upon ones personal results—which in turn would be a function of the distribution of biologically predetermined types coming into ones practice—one might (1) remain as aggressive in surgical therapy as a first year surgical resident or (2) be ready to abandon the scalpel altogether in favor of some super-voltage machine or (3) write medical articles in a lay magazine warning of the dangers of operation in the treatment of cancers.

Until there is some simple means of identifying the small but incurable lesion and avoiding non-beneficial radical operation, we should continue to use the most effective weapon at our disposal for treating cancer of the breast, that is the radical mastectomy. *J.L.S.*

1. Haagensen, C. D. and Stout, A. P., Carcinoma of the Breast, Annals of Surgery, 134: 151, 1951.

Scientific Articles

Extracervical Cancer Detection by Fluorescent Cytology— A Preliminary Report

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THOUGH not the first investigator to attempt cancer diagnosis through cytologic methods, Papanicolaou[1] in 1928 demonstrated the feasibility of cytologic cancer detection. In the ensuing years he perfected the technique into the form that is universally employed today.[2] The most intense study has been centered on the uterine cervix and from this concerted effort there has resulted a radical modification in the concept of cervical carcinoma. The mean age for detection of cervical carcinoma has been lowered to 35 years, more than a decade younger than was previously possible. The death rate from this neoplasm has been lowered over 90 per cent in areas employing mass screening programs. Cervical carcinoma may also be anticipated in advance of its occurrence by certain cytologic criteria.[3] Any of these phenomena considered alone conclusively proves the value of cytologic studies.

Other areas of the anatomy have stubbornly resisted the cytologic breakthrough. Smears and cell blocks stained by conventional techniques frequently fail to give sufficient morphologic information in early cases to enable conclusive diagnosis even in expert hands. The diagnosis of early malignancies of the central nervous system, respiratory tree, salivary glands, gastrointestinal tract, genitourinary system (other than the cervix), skin, mammary glands, and lymphatics and advanced pleural and peritoneal metastases is usually made by biopsy rather than by cytology. The explanation for the inadequacy of the cytologic method in extracervical regions is the difficulty encountered in differentiating with certainty a bizarre benign cell from a well differentiated malignant cell. Cellular appearance alone in the absence of architectural change is not enough to enable diagnosis in most instances. Some fundamental difference between benign and malignant cells had to be selected and exploited to enable differentiation. Special stains examined through conventional light

microscopes have been used extensively without notable success.[4]

von Bertalanffy[5] in 1956 succeeded in unmasking malignant change by an ingenious fluorescent analysis of intracellular ribonucleic acid (RNA) content. It had long been known that benign cells contained little RNA whereas malignant cells, even during resting and early degenerating phases contained considerably larger amounts.[4] Exploiting this characteristic through ultraviolet microscopy and fluorochrome dyes, benign cells, rich in deoxyribonucleic acid (DNA) appeared greenish; while malignant cells with increased RNA content fluoresced reddish orange. Of the various fluorochrome dyes investigated, acridine orange (hereafter referred to as "AO") gives the best results. Other investigators more recently have confirmed these observations of von Bertalanffy.[6,7] The AO method is now used routinely at Walter Reed Hospital. Although the initial work has thus far revolved around the cervix, this method of specifically labeling malignant cells has opened the way to earlier and more accurate diagnosis in other areas of the body.

Materials and Methods

This preliminary study consisted of preparations made from over 200 patients. The specimens were collected and prepared in accordance with methods outlined by Papanicolaou[2] or Solomon.[8] Every specimen was studied by the AO fluorescent technique as well as the Papanicolaou (Pap) technique except for hematologic specimens which were stained with Wright's technique. A fluorescent classification was given prior to study by the Papanicolaou method. In instances of any classification of malignancy by either technique, a biopsy was performed.

Specimens which were collected by superficial scraping or which consisted of cell-rich secretions, except those from the cervix which were intrinisically tenacious, were smeared directly on albuminized slides. When a specimen was fluid and cell-poor such as cavity fluids and urine, 15 to 60 cubic centimeters, depending on the amount of sediment, were centrifuged. It is important to process the specimen immediately without fixation. Cavity fluids frequently contain protein which will coagulate in the fixative and bind the cells. Successive portions of the fluid were added to the same centrifuge tube after decanting the supernatant material. Following the final decanting of the specimen fluid, the sides of the tube were washed down with normal saline to resuspend the cells, and the tube centrifuged once again. It has been shown[8] that the final washing step is important in that large numbers of malignant cells may remain on the side of the tube. The saline supernate was then decanted and the sediment smeared on albuminized slides. The slide was fixed in ether alcohol without delay. After fixation for 30 to 60 minutes, the slide may either be left in the fixative, or removed and dried. In the past, several objections have been raised against allowing the material to dry even after fixation, but several methods have been devised to prevent any deleterious effects, and the rehydration method of Nieburgs[9] has proven suitable for study of slides after drying in room air for periods of several weeks.

Staining for the fluorescent method was performed in accordance with the method cited previously.[4] The hydrated slide was immersed in 0.01 per cent Acridine Orange (AO) followed by two rinses in M/15 Phosphate buffer, and an interval in M/10 Calcium Chloride to enhance differentiation. The preparation was immediately scanned microscopically using an ultraviolet light source and appropriate filters. Several highly satisfactory apparatus are available commercially. The Papanicolaou technique employed was of the conventional type routinely employed in various laboratories today.[2]

Case Reports

TGS, 56-Cau-M, noted painless hematuria without other symptoms. Cytologic examination by the Papanicolaou method without concentration by the method suggested above was unsatisfactory because of lack of cellularity. Routine urinalysis showed gross hematuria. Urologic consultation was advised, but since hematuria did not recur, the patient did not seek further care until approximately two months later when hematuria was again noted. Another preparation was made employing the improved concen-

tration method. This was read as Class V by the AO method (figure 8), but only Class III (suspicious) by the Papanicolaou technique. The patient was hospitalized and three small papillomata were removed. Pathologic diagnosis: Transitional cell carcinoma, urinary bladder.

JHS, 59-Cau-M, was hospitalized with a diagnosis of Laennec's cirrhosis, right hydrocele, congestive heart failure, and arteriosclerotic heart disease with coronary insufficiency. An abdominal paracentesis was performed to relieve the ascites. The ascitic fluid prepared as described disclosed large numbers of malignant cells, Class V, by the AO technique (figure 9) and occasional cells identified as malignant, Class IV, by the papanicolaou stain (figure 10). After consultation, an abdominal exploration was performed and a liver biopsy taken. The patient subsequently expired from a massive pulmonary embolus. An autopsy was performed. Anatomic diagnosis: Primary carcinoma, liver.

HHS, 68-Cau-M, had one episode of hematuria which was painless and unassociated with other symptoms. He was seen several months later by a urologist for a check-up and gave a history of the single episode described above. The routine examination of the urine revealed only microscopic hematuria. A specimen was processed as previously outlined by the urologist in his office. By the AO method the specimen was designated Class V (figure 3. The Papanicolaou smear (figure 4) confirmed the impression when prepared by the same concentration method. Two papillomata were excised cytoscopically. Pathologic diagnosis: Transitional cell carcinoma, urinary bladder.

NC, 60-Cau-F, had a left radical mastectomy in 1952. She had consulted her family physician for symptoms described as those of a cold. She failed to improve after medication and sought the advice of an internist who found a left pleural effusion. She was hospitalized and a thoracentesis was performed. A portion of the fluid was sent to the laboratory for cytologic study, and another portion prepared by the concentration method was examined by both AO and Papanicolaou methods. The routine preparation was reported negative, but the AO and Papanicolaou methods on the concentrated material were both reported as positive, Class IV. By the Papanicolaou technique the possibility of a lymphoma was entertained. A supraclavicular node was removed for biopsy. Pathologic diagnosis: Metastatic carcinoma with sarcoid reaction.

TJ, 60-Cau-M, presented the complaint of a 2 by 3 mm irritating lesion on the dorsum of the neck. This had been treated with various creams and ointments without relief. All treatment was discontinued for a period and the lesion was scraped with a dull scalpel blade and spread on an albuminized slide. In this case the cells were separated into a thin smear with a small amount of saline and allowed to adhere to the slide by evaporation of the saline in order to avoid loss by flotation during fixation. The slide was fixed in ether-alcohol and examined cytologically. Large numbers of AO positive cells were seen (figure 5) and designated Class V, but the Papanicolaou was reported as abnormal, Class III. The lesion was excised. Pathologic diagnosis: Basal cell carcinoma (figure 6).

WY, 48-Cau-M, had persistent pulmonary symptoms and a suspicious hilar mass. Because of the progressive nature of the symptoms and a negative Papanicolaou smear of his sputum, a thoracotomy was performed and a specimen of the mediastinal lesion was removed for biopsy. Additional sputum was examined by the AO and Papanicolaou techniques and interpreted as Class V and III respectively (figure 2). This AO cytologic impression was confirmed by the biopsy report. Pathologic diagnosis: Bronchiogenic carcinoma (figure 1).

MC, 21-Cau-F, had a cervical smear taken by a gynecologist as part of a routine examination. The patient was nulliparous and had a normal appearing cervix. Both the AO and Papanicolaou methods were positive for carcinoma, Class IV (figures 15, 16A). A biopsy was performed. Pathologic diagnosis: Carcinoma of the Cervix, in situ (figure 16B).

1. Bronchogenic carcinoma, poorly differentiated.

2. AO stained sputum of same case; numerous cells of this type were seen. Other staining methods yielded equivocal results.

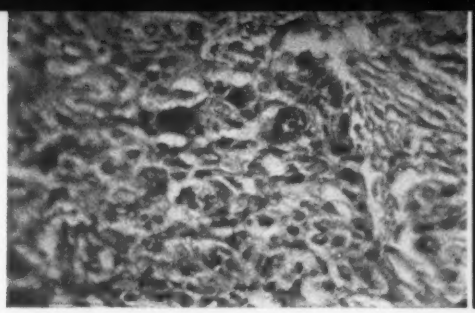


Figure 1. H & E stain x 450

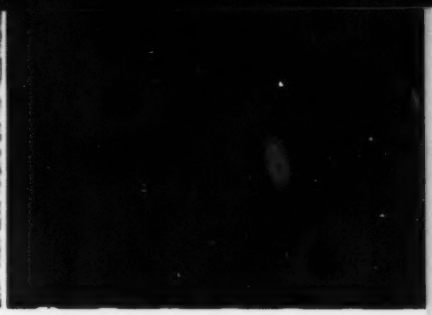
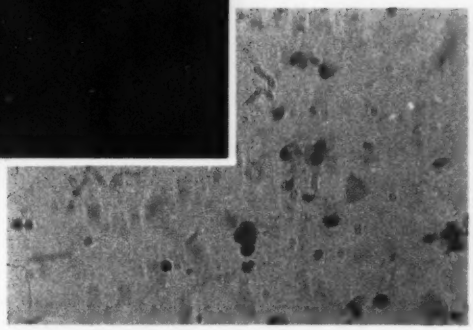


Figure 2. AO stain x 450

3. Carcinoma of bladder, urine sediment, AO stain showing striking orange-red fluorescence enabling rapid detection.

Figure 3. AO stain x 450



4. Some cells were sufficiently abnormal to permit positive diagnosis by conventional stains.

Figure 4. Papanicolaou stain x 450

5. Basal cell carcinoma of skin. AO stain showing bright orange degenerating malignant cells; conventional stains were unsatisfactory.

Figure 5. AO stain x 450

6. Same case, biopsy.

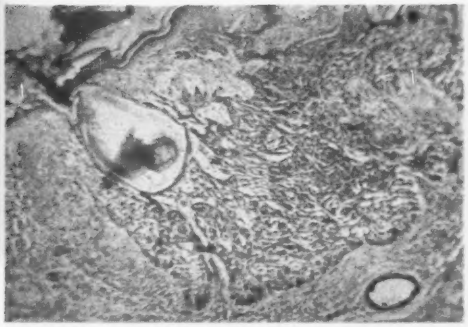


Figure 6. H & E stain x 100

7. Lymphatic leukemia, unconcentrated smear of peripheral blood. Primitive lymphocytes showing red fluorescence.

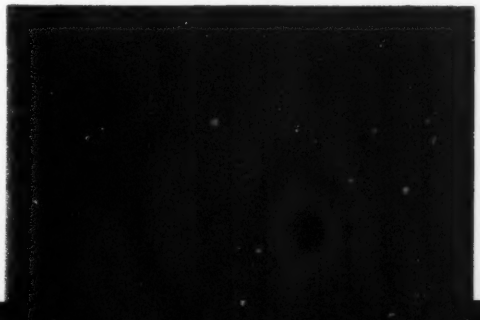


Figure 7. (left) Lymphatic leukemia x 450

Figure 8. (right) Carcinoma urinary bladder, AO stain. Pap stain of this case was class III.

Figure 9. AO stain x 450

9. Carcinoma of liver—malignant cells in ascitic fluid.

10. Papanicolaou stained preparation from same case.

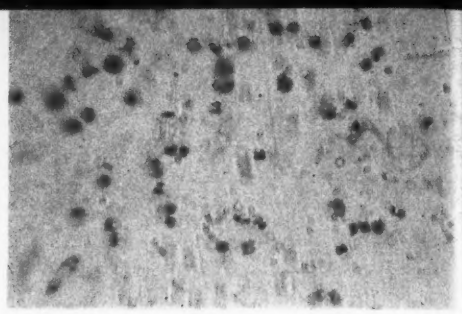


Figure 10. Pap stain x 450

1. Touch preparation from carcinoma of breast. Increased NA content of malignant cells as indicated by intense orange luorescence in the entire field.

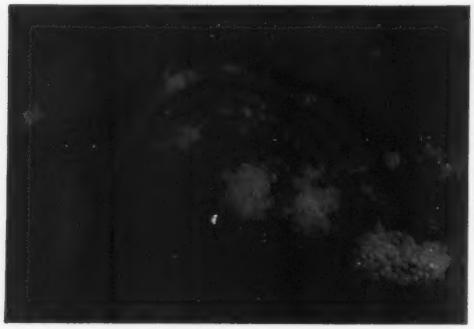


Figure 11. AO stain x 100

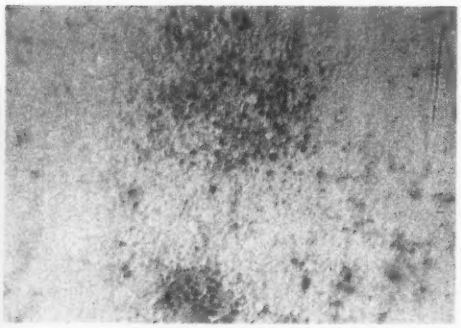


Figure 12. Pap stain x 100

12. Papanicolaou stained preparation from same case

3. Papanicolaou stained preparation containing Tri-omonads and inflammatory cells. Parasites are indi-ted by arrows.

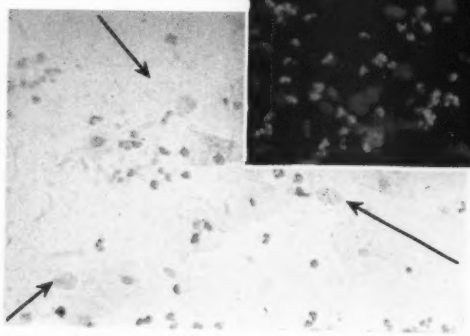


Figure 13. Pap stain x 450

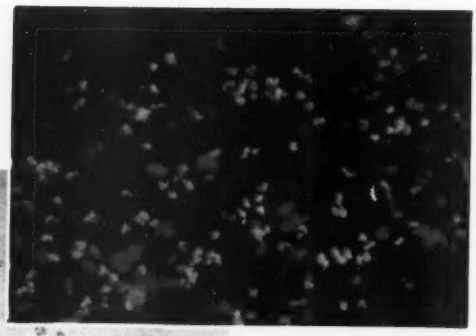
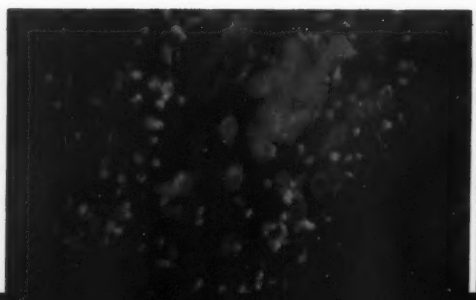
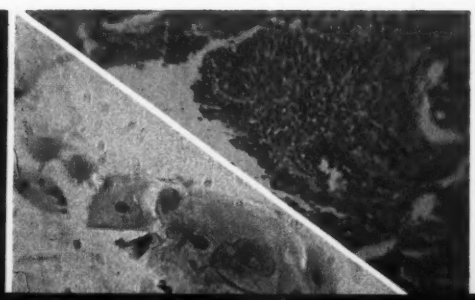


Figure 14. AO stain x 450

14. AO stained preparation from same case showing vivid red fluorescence of parasites contrasted with green inflammatory exudate.

5. & 16. Carcinoma of ervix as demonstrated y AO, Papanicolaou tain, and biopsy.





Discussion

To the present time extracervical cytologic cancer detection using conventional staining techniques has not been satisfactory. The AO fluorescent staining technique shows great promise in supplementing the purely morphological approach with a histochemical color phenomenon. Through analysis of the nucleic acid content of cells, the AO stain differentiates normal tissue from cancer tissue in a rather striking and colorful manner.

The cases cited above have confirmed the value and in some cases the superiority of the AO technique over methods heretofore available. This is especially true in the field of extracervical specimens. In addition to the high degree of sensitivity and accuracy intrinsic in the AO procedure other advantages are: (1) the method is rapid, requiring six minutes staining time vs. thirty minutes by conventional methods; (2) there is economy of expendable materials; (3) screening can be accomplished rapidly with less probability of false negative results. These and other advantages of the AO method may well provide the answer to the problems in mass screening programs for early cancer detection.

False positives with the AO techniques are occasionally encountered due to the high RNA content of some rapidly proliferating benign lesions. In these instances the Papanicolaou technique is necessary to provide a more conclusive evaluation. In all cases of malignant classification or repeated suspicious designation a specimen must be removed for biopsy for confirmation of malignancy.

The AO fluorescent technique shows promise in other fields besides tumor detection. Preliminary work in this laboratory indicates that primitive white cells contain increased RNA and fluoresce a brilliant red, while mature white cells show green fluorescence. This may afford a simple reliable means by which normal white cell constituents of the blood may be differentiated from their malignant counterparts (figure 7). It is possible that tumor cells may be identified more readily in plasma concentrates by this method than by conventional techniques. Through their striking red fluorescence bacteria, fungi, and protozoa (figure 14) stand out vividly in smeared preparations. This may augment their detection and identification in certain body fluids or excreta. Inclusions in cytomegalic cells stand out clearly as brilliant red bodies. It has been observed in this laboratory that intracellular viral plaques will show red fluorescence.

It is established[10,11] that a direct correlation exists between RNA content and radiosensitivity. It is anticipated that the AO fluorescent method of tumor cell analysis may shed light on the degree of tumor sensitivity to radiation therapy.

The wider use of this technique may facilitate advances in the early diagnosis of extracervical lesions comparable to those effected by the Papanicolaou method in uterine cancer.

Summary

Six cases of extracervical malignancy detected by a fluorochrome fluorescent technique are presented. This new method of cancer detection seems at the present time to offer greater reliability and enables earlier detection of malignancies than is realized by conventional cytologic techniques. Its application in the fields of bacteriology, mycology, virology, hematology, parasitology, and radiology are cited briefly.

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Despite the current popularity of diuretic therapy, digitalis administration is still the most effective measure in treatment of congestive heart failure.

Digitalis Therapy in Congestive Heart Failure

This is the fourth of a series of articles prepared for *The Journal* under the auspices of the Oklahoma Heart Association and its committee on Professional Education, emphasizing the theme of the Association for the current year.

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CONGESTIVE HEART FAILURE results from an imbalance between cardiac muscle strength and the work load imposed upon the heart, that is, the heart is unable to pump enough blood to supply the metabolic needs of the body. With few exceptions, the essential defect in congestive failure is a deficient cardiac output resulting from impaired myocardial function. The muscle fibers have usually been subjected to increasing tension in diastole or to excessive resistance to contraction, eventually leading to overstretching and fatigue of the myocardium and impairment of its contractility. The resulting deficiency in cardiac output triggers certain renal and hormonal mechanisms which result in the retention of sodium and water, expansion of extracellular fluid volume, edema, visceral congestion, hypoxia, etc., all of which further compromise myocardial function.[1-4]

Logically, the objectives of therapy in congestive failure are improvement of myocardial function and cardiac output, and reduction of the work load placed on the heart. The digitalis glycosides are the only pharmacologic agents which possess direct cardiotonic properties, and their chief therapeutic value lies in the ability to increase a deficient cardiac output. The various other measures used in congestive failure have no direct action on cardiac muscle, but indirectly improve the clinical state by reducing the work load of the heart, e.g., physical rest, salt restriction and diuretic therapy, aspiration of large transudates, venesection, etc.

During recent years, digitalis therapy has often been neglected in favor of measures designed to mobilize and excrete extracellular fluid accumulations. In the earlier stages of congestive failure, physical rest, digitalis therapy and perhaps moderate restriction of salt intake are usually sufficient to restore compensation. Digitalis therapy alone may not be adequate in the more advanced stages of failure particularly if there is severe myocardial damage, and other measures must be instituted to aid in reduction of the work load of the heart to a degree compatible with the functional capacity of the myocardium.

The Action of Digitalis

When therapeutic doses of digitalis are given in congestive failure, the following events occur: the ventricular rate usually slows, cardiac output improves, right atrial and peripheral venous pressures fall, pulmonary arterial diastolic pressure falls, arteriovenous oxygen difference decreases, and diuresis of salt and water occurs.[5, 6] It is difficult to decide which of these closely interrelated cardiac, renal and vascular phenomena are primary and which are secondary effects of digitalis activity. The present concensus is that all of these events are the result of the improved myocardial function and cardiac output induced by the direct cardiotonic action of digitalis.

In low-output failure digitalis improves myocardial contractility and increases cardiac output by increasing the force of systolic contraction;[7] the duration of diastole is lengthened, thus permitting greater time for myocardial rest and ventricular filling;[8] the ventricles empty more completely, thereby increasing stroke volume. The diastolic size of the heart is reduced by digitalis,[9] and since oxygen consumption is a function of initial diastolic fiber length,[10] the oxygen expenditure for any work performed by the heart muscle is reduced.

The biochemical effects of digitalis on the heart muscle are not entirely clear at the present time. Certain in vitro observations[11] indicate that digitalis is concentrated near the contractile elements, myosin and actin, rather than in the mitochondria which harbor the glycolytic enzymes utilized in the Krebs cycle. Therapeutic doses of digitalis have been shown to increase the ionic concentration of potassium in the muscle cell, while toxic doses of digitalis appear to cause potassium ions to be released from the cell.[12] According to the views of Szent-Gyorgyi,[13] the contractile elements of the cell, myosin and actin, are prevented from uniting by the ionic concentration of potassium in the cell. When excited by a stimulus, the cell membrane is depolarized and becomes permeable to potassium ions which are allowed to escape from the cell, thus permitting the union and subsequent spiral folding of actin and myosin. Studies of myocardial metabolism in low-output failure reveal no deficit in oxygen utilization,[14] no reduction in the supply of the energy donor adenosine triphosphate (ATP),[15] and no change in the consumption of catabolic substrates such as lactate or pyruvate.[16] These observations lead to the conclusion that the essential metabolic defect in low-output failure does not lie within the phase of energy production, but rather in the lack of efficient energy utilization on the part of the overly stretched contractile elements of the heart muscle. Digitalis apparently acts directly on the contractile elements of the heart muscle. Digitalis apparently acts directly on the contractile elements of the cell, enabling them to use the normally available energy supply more efficiently, perhaps through the effects of the drug on the concentration of potassium in the cell.

Digitalis preparations possess one important extracardiac action; namely, the vagal action of small doses of these drugs in atrial fibrillation which will slow the ventricular rate. At this point exercise or atropine administration will cause exaggerated acceleration of the ventricular rate. Full therapeutic doses of digitalis slow the ventricles by directly depressing the arterioventricular conduction tissue and this effect cannot be erased by exercise or atropine. This effect is useful as an indication of full digitalization in the patient with atrial fibrillation by observing the response of the ventricular rate to exercise.[17] The slowing of the heart, which often occurs when digitalis is administered to the patient in congestive failure with a sinus rhythm, is apparently not the result of the vagal action or of depression of atrioventricular conduction, but is the result of the overall improvement in myocardial function. Measurable clinical improvement is often observed in such patients prior to any significant decrease in the ventricular rate. In such patients the heart rate does not serve as a reliable guide to the level of digitalis effect. Toxic doses of digitalis sometimes induce arrhythmias with rapid ventricular rates simulating atrial fibrillation or sinus tachycardia,[18] and further efforts to slow the heart rate with digitalis can prove to be disastrous.

Some rather characteristic electrocardiographic changes may be induced by digitalis administration: the QT interval may be shortened; a rather concave depression may appear in the RST segment often associated with inversion of the first portion of the T wave; the PR interval may be lengthened due to atrioventricular depression, and varying degrees of heart block may result from toxic doses of the drug. Excessive doses of digitalis can cause practically any of the arrhythmias from premature contractions to ventricular fibrillation. Lown and Levine have emphasized the importance of recognizing paroxysmal atrial tachycardia with varying degrees of AV block, since it is almost always caused by excessive doses of digitalis, and because the arrhythmia is often mistaken at the bedside for atrial fibrillation, which may lead the physician to give more digitalis in the hope of slowing the ventricular rate.

The Various Cardioactive Preparations

The cardiotonic properties of the various preparations of digitalis depend upon their content of one or more of the cardiac glycosides. All of the digitalis glycosides are structurally similar in having a steroid nucleus with a lactone ring at C-17, and a complex sugar moiety at C-3. Differences in the pharmacologic properties of the various glycosides are due to differences in the structure of the sugar group and to the nature and position of other substituent groups.[19, 20]

There are many commercial preparations of digitalis available under a bewildering variety of proprietary names. No useful purpose is served by the attempt to remember all of the different commercial preparations. A more rational grouping is formed by the generic classification of the cardioactive drugs. There are four naturally occurring parent substances, each containing one or more of the currently used cardiac glycosides: Digitalis purpurea, Digitalis lanata, Strophanthus gratus and Strophanthus kombe.

Digitalis purpurea is the only whole plant substance now used in the crude form. Digitalis USP consists of a mixture of natural active glycosides with inert material. Standardization is accomplished by determination of the lethal dose in pigeons, one USP unit being equivalent to 0.1 Gm. of USP Digitalis Reference Standard. Commercial USP preparations must have a potency of no less than 85 per cent nor more than 120 per cent of the reference standard.

Digitoxin is derived from a glycoside of Digitalis purpurea by removal of a glucose. It may also be obtained from lanatoside A, which is one of the glycosides of Digitalis lanata, by removal of glucose and acetyl. Digitoxin USP is standardized by a chemical method which measures the content of active glycosides. This method also measures the relatively biologically inert substance gitoxin as though it were digitoxin, and significant quantities of this material may be found in many commercial preparations.

Gitalin, amorphous is derived from Digitalis purpurea by a special method of extraction and purification, and contains a mixture of natural glycosides. As yet there are no legal methods of standardization.

Digitalis lanata is the parent plant of several cardioactive substances, although it is not itself available in the crude form for clinical use.

Digilanid is a purified mixture of the three natural glycosides of Digitalis lanata, lanatosides A, B and C, and these substances are present in approximately the same proportions as they occur in the plant.

Lanatosides A and B are not available, as such, for clinical use.

Digitoxin is derived from lanatoside A, and acetyl digitoxin is also derived from lanatoside A by the removal of glucose, but not of acetyl.

Lanatoside C is a purified glycoside of Digitalis lanata. The USP products are standardized by the same method of pigeon assay as used for digitalis leaf. Digoxin, which is very similar in action to Lanatoside C, is derived from that glycoside by removal of glucose and acetyl. Standardization is accomplished by colorimetric methods, and USP Digoxin must assay to a potency of not less than 90 per cent nor more than 110 per cent of the USP Digoxin Reference Standard.

Strophanthus gratus and the wood of the ouabao tree are the sources of ouabain, which is prepared for intravenous use only, and is standardized by the pigeon assay method.

Strophanthin K is a mixture of three or more natural glycosides of Strophanthus kombe, and is standardized, according to the N. F. IX, to one-half the potency per milligram of Ouabain USP.

Acetyl strophanthidin is a recently developed synthetic preparation derived from the aglucone strophanthidin, and cannot truly be classified as a glycoside. The drug is assayed in cat units against a reference standard, and has a potency of approximately three cat units per 0.5 mg. of drug.

Quantitative Differences in the Various Cardioactive Preparations

Although the biologic effect of all cardiac glycosides are essentially alike, significant quantitative differences exist in the degree and uniformity of gastrointestinal absorption, and in the rates of onset and decline of biologic effects. These differences influence the choice of a preparation for clinical use.

Preparations of digitalis for oral administration must be adequately and uniformly absorbed from the gastrointestinal tract. The strophanthus derivatives are so poorly and erratically absorbed as to be unsuitable for oral use. Lanatoside C is absorbed to the extent of about 10 per cent of the oral dose,[21] and is generally considered a poor choice for oral administration. The other digitalis preparations are adequately and uniformly absorbed from the gastrointestinal tract as follows: digitalis leaf—20 per cent, digoxin—50 per cent, acetyl digitoxin —67 per cent, and digitoxin—virtually 100 per cent.[8, 22]

The various digitalis glyosides differ widely in the rates of onset and decline of biologic activity,[24] even when given by the same route in therapeutically equivalent doses. Very little is known of the processes of degradation or excretion of the glycosides with the possible exception of digitoxin. Digitoxin is adsorbed by plasma albumin[23] which may be responsible for the delay in its onset of action and its prolonged effect. The strophanthus derivatives, when given intravenously, exert a full therapeutic effect within a few minutes to one hour. Characteristically, these rapidly acting preparations produce effects which are rapidly dissipated, making them unsuitable for long-term maintenance of digitalis effect. Lanatoside C and digoxin, when given intravenously, produce some effect on the heart within a few minutes, and generally exert a maximal effect within three hours, which declines over a period of 48 to 72 hours. Digitoxin and digitalis leaf are similar in their rates of onset and decline of activity. Digitoxin may act slightly faster when given intravenously; however, the time differential is not great. When digoxin is administered orally, its full effects are not apparent for about six hours, although its rate of decline is the same as when given intravenously.

The Clinical Use of the Digitalis Preparations

As previously stated, the principal therapeutic value of digitalis in congestive failure lies in the ability to increase a deficient cardiac output through improvement of myocardial function. The effectiveness of digitalis in a given case is determined largely by the nature of the underlying process leading to the development of failure. Digitalis may actually decrease the output of a normal heart, hence, the drug has no value in the treatment of dyspnea or edema of non-cardiac origin. When digitalis is given to the patient with an enlarged, but otherwise well-compensated heart, the cardiac output may also be decreased.[25] Christian[26] once advocated the "prophylactic digitalization" of such patients; however, this practice is not widely accepted at the present time. Digitalis therapy may be distinctly beneficial for the patient in incipient failure, that is, the cardiac output is sufficient except for brief periods when the demands of the body for blood exceed the reserve capacity of the myocardium. The physician can often find no signs of failure in such patients at the time of examination, although occasionally a gallop rhythm or an increased circulation time may be found.

Digitalis therapy is most effective in the patient with low-output failure which, fortunately, is the usual type of failure resulting from hypertensive, arteriosclerotic or

valvular heart disease. The patient with tight mitral stenosis and a sinus rhythm may respond poorly to digitalis administration,[18] since the pulmonary congestion is often the result of a compensated right ventricle continuing to force the blood into the pulmonary circuit which is already overloaded by blood dammed back by the tight mitral obstruction. Relief is often marked in such patients following the onset of atrial fibrillation and/or right ventricular failure. Repeated myocardial infarctions may reduce the total cardiac muscle mass to the point where the total potential contractile powers of the myocardium are below the level which would maintain a satisfactory cardiac output even with full digitalization. Digitalis therapy should be used in such patients, but the results may be disappointing.

When congestive failure is caused by constrictive pericarditis, acute myocarditis, or cyanotic congenital heart disease, digitalis administration is not particularly effective, although its use is not contraindicated in these situations.[18]

So-called high-output failure does not usually respond well to digitalis therapy, unless there is some associated intrinsic heart disease.[24] Thyrotoxicosis, severe anemia, severe infections, arteriovenous fistulas, beri-beri, pulmonary disorders, or pregnancy are all capable of producing this syndrome. The mechanism of failure in such cases is apparently the decreased peripheral vascular resistance which permits massive amounts of blood to be diverted through low resistance vascular channels into the venous circulation, by-passing the capillary bed and the kidneys. The venous return to the heart results in an increased load which is augmented further by renal retention of salt and water. The treatment of high-output failure should be aimed primarily at the underlying cause; however, digitalis may be given particularly if the situation is associated with intrinsic heart disease.

The Administration of Digitalis Preparations

Having made the decision to digitalize a patient in congestive failure, the selection of a suitable drug must be based upon the individual needs of the case. The urgency of the clinical situation must be assessed, i.e., a case of severe pulmonary edema may require rapid intravenous digitalization, whereas the average case can usually be digitalized by the oral route at a more leisurely rate with one of the longer acting preparations. The patient who is unable to take oral medications must be digitalized intravenously. In addition, every effort must be made to discover whether or not the patient has recently received a digitalis preparation. Too often digitalis intoxication results from neglecting to obtain this information when the patient is transferred from one physician to another.

Practicing physicians are interested in utilizing dosage schedules for drugs which can be applied safely to a majority of patients. The digitalis preparations cannot be administered effectively according to any rigid dosage schedules, but rather each patient represents an individual problem in the bio-assay of digitalis. Despite this fact, certain average doses of the cardioactive drugs have been determined which are usually safe to follow as guides for administration to the individual patient.[24] Digitalis and digitoxin are the drugs most frequently used for oral administration. Both preparations are very similar in producing effects which are slowly dissipated and therefore held to achieve a stability of effect. Minor gastrointestinal toxic symptoms appeared in less than three per cent of a group of 512 patients who had each received a single dose of 1.2 mg. of digitoxin. Single doses of 2.0 mg. of the drug induced toxic symptoms in nearly one-third of a group of 98 patients.[27] A few patients may be fully digitalized with as little as 1.0 mg. of digitoxin, while a rare patient may require 3.0 mg. of the drug to reach a full therapeutic effect. The average digitalizing dose of digitalis leaf is approximately ten times that of digitoxin. Acetyl digitoxin will accomplish full digitalization in the average patient with 2.0 mg.; however, the range of dosage varies from 1.5 to 2.5 mg. Batterman, De graff and Rose[28] have claimed that gitalin has a much wider therapeutic ratio than other digitalis preparations; however, others[29] have failed to confirm this point. It has been observed that gitalin is apt to induce cardiotoxic manifestations without causing gastrointestinal

symptoms. The average digitalizing dose of gitalin is 5.0 mg., with a range of 4.0 to 8.0 mg. Digoxin is more often reserved for intravenous administration; however, it may be given satisfactorily by mouth. The average oral dose of digoxin required for full digitalization is about 3.0 mg. with a range from 1.0 to 4.0 mg.

The previously-listed average digitalizing doses are generally safe to use as guides, but should not be given as single doses. The best plan of digitalization in the average patient is to administer the chosen drug in small divided doses at sufficiently long intervals to allow observation of the full effect of each dose. Full digitalization can usually be accomplished within 24 to 72 hours by this method. Using digitoxin, a satisfactory plan might be as follows: 0.4 mg. of the drug is given every eight hours for three doses, observing the clinical effect of each dose. If full digitalization has not been achieved with 1.2 mg. of the drug, subsequent smaller doses of 0.2 mg. of digitoxin may be given every eight hours until the desired effect is reached. A similar technique may be followed using therapeutically equivalent doses of any of the other oral preparations of digitalis.

Seldom is the end-point of full digitalization sharply defined in congestive failure, except when there is associated atrial fibrillation. Some physicians prefer to digitalize the patient to a mildly toxic state; however, some patients object strenuously to the toxic symptoms, and also certain seriously-ill patients have such a narrow margin between optimal digitalization and toxicity that excessive doses of the drug may be dangerous. Observation of the electrocardiographic effects of digitalis cannot be relied upon to indicate either the level of optimal digitalization or the nearness of intoxication. Once toxicity has developed, the electrocardiogram may sometimes help to make the proper diagnosis by showing some characteristic conduction defect or arrhythmia. Some patients are so critically ill that the end-point of digitalization may be exceedingly difficult to estimate; indeed, it may be virtually impossible to decide whether the patient is intoxicated or whether additional amounts of digitalis are needed. Unless the situation is extremely urgent, the wisest course is to discontinue the drug temporarily while observing whether the patient's condition improves or worsens. Small intravenous doses of the rapidly dissipated glycosides have been given to such patients in the hope of clarifying the situation either by aggravation of toxic symptoms or by improvement of the state of failure. This procedure is hazardous, but may prove to be useful in a few selected cases. The author prefers to use lanatoside C or digoxin for this purpose. Lown and Levine[18] have reported similar use of the ultra-short acting drug, acetyl strophanthidin for such cases; however, fatalities have been reported from this practice,[30] and the use of this drug should probably be limited to purely investigative studies in highly-trained hands.

Upon reaching the desired level of digitalization, the physician must then determine the daily dose of drug necessary to maintain an optimal digitalis effect. This again is a clinical experiment in which an average dose of the chosen drug is given on a trial basis over a period of time sufficiently long to allow errors in dosage to become apparent. Several weeks may elapse before errors in dosage of the longer-acting preparation are evident. Average maintenance doses of the oral digitalis preparations are approximately as follows:[24] digitalis leaf—0.15 Gm., digitoxin—0.15 mg., acetyl digitoxin—0.3 mg., gitalin — 0.5 mg., digoxin — 0.5 mg. The range of maintenance dosage of each drug varies widely from patient to patient, and the physician must titrate each patient clinically to the correct dose.

Several digitalis preparations may be given intravenously to patients who are unable to receive oral medication. Except in urgent situations, the same general plan of administration of each dose should be followed as when digitalizing a patient orally; that is, small divided doses of the drug at intervals long enough to permit observation of the effect. The intravenous dose of digitoxin is the same as the oral dose; however, the other digitalis glycosides are given in considerably smaller doses by the intravenous route. The average intravenous digitalizing dose of acetyl digitoxin is 1.5 mg., and for digoxin is 1.0 mg. Lanatoside C is also

a satisfactory intravenous preparation, the average digitalizing dose being 1.6 mg. It is usually advisable not to attempt full digitalization by the intravenous route in less than 48 hours.

Rapid intravenous digitalization, although hazardous, may be required as a life-saving measure in certain cases such as severe pulmonary edema. Digoxin or lanatoside C are more frequently used for this purpose than the other glycosides. Both drugs induce a clinical effect within a few minutes and maintenance of the effect is more stable than with the strophanthus derivatives. Kay[24] has recommended the following plan for rapid intravenous digitalization with digoxin or lanatoside C: one-half of the average digitalizing dose is given slowly by vein over a period of five minutes, followed, if necessary, by half as much in each of two hourly doses. Subsequent doses should be withheld for six hours to allow full development of the drug effect. Ouabain produces a full therapeutic effect much more rapidly than digoxin or lanatoside C. The absolute maximum dosage for ouabain is as follows: an initial dose of 0.5 mg. to be followed every 30 minutes with 0.1 mg. of ouabain for no more than three doses. Once the patient has passed the critical period, further digitalization should be carried out at a slower rate, preferably by mouth.

Digitalis Intoxication

Digitalis intoxication has allegedly become more frequent in recent years, perhaps because of the more widespread use of digitalis and probably because of the trend toward administration of larger doses over shorter periods of time. The common practice of using potent diuretic drugs which cause increased excretion of potassium from the body may also contribute to the increased incidence of digitalis intoxication.

The most common and usually the earliest manifestations of digitalis intoxication are gastrointestinal symptoms of anorexia, nausea, vomiting or diarrhea. If such symptoms occur within one or two hours after oral administration of the drug, the effect is one of local gastrointestinal irritation, otherwise such effects are due to involvement of the central nervous system. Visual disturbances, restlessness, headache, mental confusion or stupor may result from the toxic effects of digitalis on the central nervous system. Digitalis intoxication also affects the heart, being capable of inducing any of the arrhythmias. Generally, the first manifestation of cardiotoxicity to be observed at the bedside is the appearance of premature contractions, often occuring in a bigeminal rhythm (coupling). Sometimes, however, premature contractions are present prior to digitalization, and may be decreased in number or completely abolished by restoring the heart to a compensated state.

It is known that calcium and digitalis have synergistic effects on the myocardium; in fact, intravenous administration of calcium salts to the digitalized patient is dangerous and has been known to have lethal consequences.[31] Recently Guber and Kallman[32] have suggested that a rational approach to the treatment of severe digitalis intoxication might include methods for lowering the serum concentration of calcium. These investigators advocate the use of the chelating agent, disodium versenate (disodium ethylene diamine tetracetate) to lower the serum calcium level. The drug is administered slowly intravenously, 600 mg. in 1000 ml. of 5 per cent glucose in water.

Frequently a patient who has appeared to be properly digitalized will develop signs of toxicity after receiving a potent diuretic. Formerly, it was believed that this phenomenon was due to redigitalization by the mobilization of edema fluid containing excessive amounts of digitalis. Subsequently it has been shown that only negligible amounts of digitalis can be identified in edema fluid, and apparently the reason for the appearance of toxic symptoms is the excessive loss of potassium ion occurring with the excretion of sodium and water. When digitalis intoxication has been induced by diuresis, the symptoms may be more rapidly improved by administration of 5.0 to 7.5 Gm. of potassium chloride orally in divided doses over a 24-hour period, unless the symptoms are severe enough, in which case the above

amounts of potassium chloride may be given intravenously in 500 ml. of 5 per cent glucose in distilled water.

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CHRISTIAN, *n.* One who believes that the New Testament is a divinely inspired book admirably suited to the spiritual needs of his neighbor. One who follows the teachings of Christ in so far as they are not inconsistent with a life of sin.

From the Devil's Dictionary by Ambrose Bierce
—Sagamore Press, Inc.

The Metabolic Response to Trauma

JAMES D. FUNNELL

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MANY INVESTIGATORS have tried to relate the metabolic changes following trauma to endocrine secretion. Most often mentioned are the secretions of the adrenal and pituitary glands.

Claude Bernard,[1] then W. Cannon[2] were the first to call attention to this. More recently Hans Seyle[3] added to this idea a concept of systemic response and an interrelationship of physiological and hormone systems. Ramey[4] has discussed the interrelationship of the sympathetic nervous system and the adrenal cortex. Moore[5] has emphasized the metabolic response to trauma, and Dunphy and Edwards[6] have stressed the importance of the metabolic response at the cellular level.

Figures 1 and 2 show some of the more important and generally accepted concepts of hormonal interrelation. The basic concept introduced by Hill[7] and elaborated by Stanley,[8] has been broadened further to include newer concepts such as the Adipokinetic factor. The purpose of this paper is to correlate these concepts and interrelationships with Moore's[5] four phases of surgical convalescence and the important changes associated with each of these phases. Finally, the therapeutic measures employed in each convalescent phase are described.

Figure 1 is based on an interlocking triad of epinephrine, neuroendocrine and a histamine-like response. When survival is threatened by insult or trauma, e.g., surgery, an epinephrine or adrenergic response is initiated first in this "flight or fight" response. Several important events occur. There is vasoconstriction, and an increase in pulse and blood pressure. Liver glycogen is released with production of a hyperglycemia, the spleen contracts, white blood cells are mobilized and fibrinogen is released. The blood sugar increase acts on receptor cells in the islets of Langerhans with insulin release and lowering of blood sugar. The decreased blood sugar further stimulates the adrenal medulla, releasing a histamine-like substance. The histamine-like response acts to cause vasodilitation and decreased blood pressure, a further stimulant to epinephrine production, and a potent stimulant of the pituitary gland.

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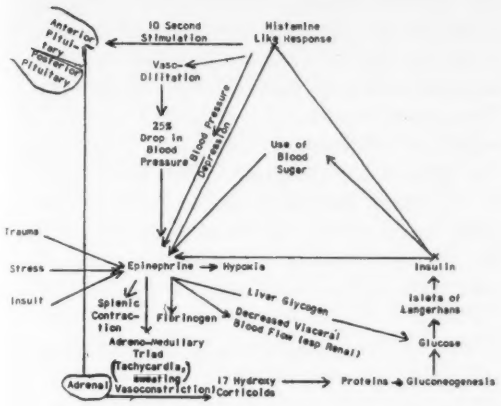

Figure 1

The pituitary figures prominently in the release from the neurohypophysis of the antidiuretic hormone (pitressin) which acts on the distal renal tubules to conserve water despite the need to excrete it. Also from the adenohypophysis come the trophic hormones. Especially important in the stress response are the adreno-cortico-trophic hormone (ACTH), somatotrophin (growth hormone), thyroid stimulating hormone (TSH), and the gonadotropins (follicle stimulatings, leutinizing and lactogenic hormones). A newly recognized hormone adipokinetic factor, concerned with mobilization of fat is of importance as a source of energy in a traumatized individual. See figure 2.

Following physical trauma ACTH acts on the middle portion of the adrenal cortex (Zona Fasciculata) with subsequent release

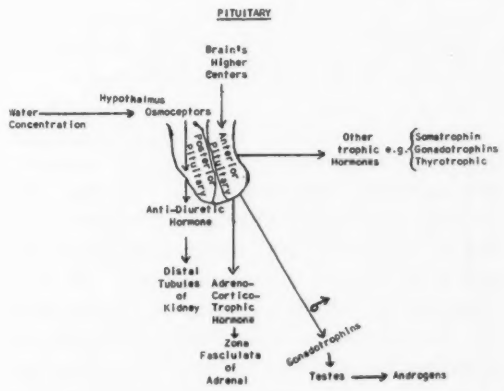

Figure 2

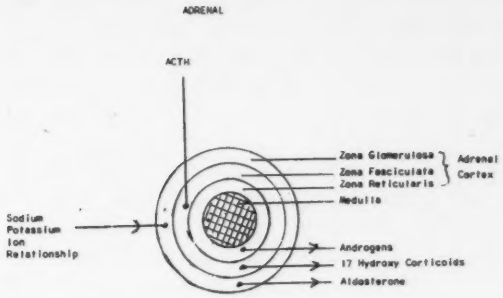

Figure 3

of Glucocorticoids (17 Hydroxy corticoids, especially Hydrocortisone). The glucocorticoids act on body proteins and by gluconeogenisis produce glucose which in turn stimulates the islets of Langerhans to release more insulin.

The sodium and potassium ions act more directly on the adrenal cortex outer layer (Zona glomerulosa) to release mineral corticoids (electrocorticoids). The most active of these, aldosterone, acts on the distal renal tubule to conserve sodium and excrete potassium.

Androgens, produced in the adrenals and testes in the male, are related to protein anabolism. In the female, estrogens are released from ovaries. See figure 3.

Other important concepts, depicted in figures 1 and 2, entail such things as the hypothamus and the neuro-endocrine feedback mechanisms as well as osmoreceptors concerned with regulation of the antidiuretic hormone. Lastly, somatotrophin may be concerned with Adipokinetic activity. Thyrotrophic hormone and the thyroid hormone regulate metabolic rate, oxygen consumption, cell division, red blood cell maturation and synergism with glucocorticoids.

Moore[5] found that four phases appeared in sequence in normal post traumatic metabolism. He called these: (1) Phase I or the adrenergic-corticoid phase, lasting an average of four days; (2) Phase II, the corticoid withdrawal phase, lasting three or four days; (3) Phase III, the spontaneous anabolic phase lasting about 20 days; and (4) Phase IV, the fat gain phase, lasting

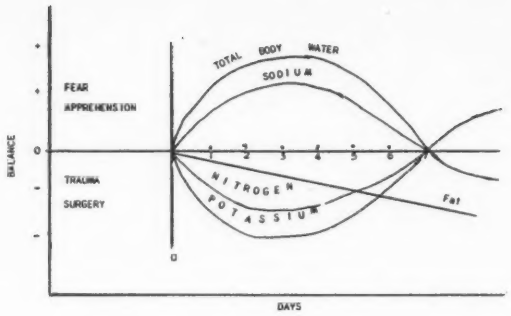

Figure 4

weeks or months. In this scheme, we enter Phase I with the neurologic stress of an approaching operation and the person at "Zero" balance for water, nitrogen, sodium, potassium and weight. Phase IV ends when balance following operation is restored again and fat and weight are re-established. During each of these phases specific events occur in the individual. By applying the basic information outlined in the figure it is possible to treat the surgical patient more accurately. For this reason let us examine each phase separately and outline some of the salient features. In the first phase, the corticoid phase, the following events occur. See figure 4.

(1) In general there is antidiuresis where water is resorbed by the renal tube. In addition sodium is retained while potassium is lost. The chief factors stimulating this are the antidiuretic hormone and Aldosterone. Other factors include decreased renal blood flow with decreased glomerular filtration as part of the adrenergic response.

(2) Changes in capillary permeability and other factors allow dilution and expansion of the extravascular component. There is also continued loss of insensible water and loss of fluid from other extra renal sources. In the latter, great importance must be placed on body fluids lost by gastric suction, vomiting, ascitic fluid, or wound and inflammatory drainage.

(3) Nitrogen balance is negative, primarily because of decreased or absence of intake. Other losses occur as amino acids are converted by gulconeogenesis, forming blood sugar. In this stage there seems to be wound priority for amino acids, especially these containing sulfur (e.g. Methionine). These are utilized in wound repair. The negative nitrogen balance has been called the Phase of Obligatory Catabolism because it is almost independent of attempts to reverse it. Abbott[9] has shown, however, that early parenteral or oral alimentation may minimize this catabolic phase.

(4) In this phase the normal potassium to nitrogen excretion ratio of 2.7:1 is altered and may become 5:1 or 6:1 or greater. This probably relates to catabolism of intracellular protein. The loss of intracellular water and potassium occurs prior to the catabolic cellular breakdown. It should be remembered that potassium is maintained intracellularly with glucose and glycogen. With the metabolism of these glycogen stores, the intracellular potassium necessary for the storage in cells migrates to the extracellular compartment and is excreted by the kidneys.

(5) There is a fall in the circulating eosinophile count. In major operations it often falls to zero and its backswing marks the beginning of the Corticoid withdrawal phase.

(6) These include responses such as adynamic ileus, lack of oral intake, a rise in body temperature, and the beginning utilization of body fat. Sex function is lost and amenorrhea occurs in the female.

The principles of treatment of surgical patients during this phase follows logically.

Phase I: During Phase I, as well as all other phases, blood loss must be replaced immediately and in adequate quantity. The problems of correct typing and cross matching as well as the danger of transfusion reaction cannot be overemphasized. The importance of rapid blood loss replacement lies not only in hemodynamics and oxygen carriage but also in the following: (1) over 90% of CO_2 carriage is by the red blood cell; (2) the effect of nitrogen depletion due to

blood loss and (3) much of the buffer capacity of the blood lies in the red blood cells.

Replacement of fluid other than blood is by quantity and kind. It is useful to remember the body is 50 to 70% water and of this approximately 40% of body weight may be considered intracellular, 15% extravascular and 5% intravascular. Important in evaluting fluid replacement is the technique of weighing the patient on scales sensitive to 20-50 gm. and replacement of loss by weight. This technique is excellent but one must remember that post-operatively fats are mobilized by the adipokinetic factor, causing a 150 to 500 gm. fat loss daily. Daily fat loss and production of metabolic water (150-250 cc) should be considered in the interpretation of weight changes, and in fluid therapy.

In addition the body continues to lose insensible water. The minimal rate for this is 750-1000 cc. per day. Minimal urine output is about 600 cc. per day though 1200-1500 cc. of urine per day is more nearly optimal. All fluid losses must be replaced parenterally or orally. In addition to replacing insensible and urinary fluid loss, extra renal losses must be replaced in kind. When small quantities of such fluids are lost by gastric suction, through fistulae, etc., their composition may be considered equal to plasma electrolyte concentrations. When larger quantities of fluid are lost electrolyte analysis should be performed on aliquots as a further guide for therapy. Body weight, volumetric or gravimetric measurements of dressings and fluids lost also are valuable adjuvents in planning replacement therapy. Because of sodium retention and, in the case of moderate extra renal losses, 5% glucose in water is usually sufficient to provide water replacement.

In Phase I, day 1, potassium usually is not given due to the forementioned endogenous sources from intracellular stores, and because of ease of oral administration on the third or fourth day. In other more severe cases potassium is given in terms of losses, 20-40 meq. per day are added to intravenous solutions. Potassium is never given when renal flow is less than 30 ml/hr, because of the danger of hyperpotassmia. Due to atonia of the gut during this phase, all administration of fluids is carried out by intravenous routes. However, sodium should be given for specific indication and not routinely during this Phase because both mineralo corticoids and glucocorticoids cause sodium and chloride retention. Sodium blood levels may show a relative decrease to 130-135 mEq/L due to an increase in total body water caused by antidiuretic hormone.

Nitrogen is in negative balance due to the catabolic phase. Endogenous utilization causes a loss of muscle mass with concurrent loss of intracellular potassium and water. Replacement therapy is relatively unsuccessful due to several factors. The first factor is energy of utilization, or energy from endogenous sources necessary to convert protein into a utilizable form. This accounts for 25% loss of protein in its original energy value. Secondly, there exists a nitrogen to calorie, or N:C, ratio which requires approximately 300 calories for each gram of nitrogen. A calorie intake below this level results in protein source consumption to meet daily metabolic needs for calories. At this stage, the wound also has priority regardless of other body needs.

A useful index for following patient progress through this stage is the eosinophile count which drops to or near "0"% following trauma (surgery). It remains low through Phase I, then swings back, often overshooting normal blood count levels. In this phase, as in later phases, fever and wound infection must be guarded against. A 1°+ rise in temperature the first two-four days postoperatively is normal.

It is important to recognize that survival depends upon adrenal production of the glucocorticoids. In patients who have received exogenous corticoid (e.g. cortisone) prior to trauma, there may be an atrophy of the adrenals as in Addison's Disease. The patient must be maintained on exogenous corticoids, operatively and postoperatively. Whenever there is a history of steroid therapy, intravenous hydrocortisone should be available in the operating room. It should

be administered immediately if there is hypotension, as this rapidly may become irreversible. However, steroid therapy will not compensate for blood loss. This must be replaced by blood.

Schlegel[10] has pointed out that the ratio of solids and water from exogenous and endogenous sources must equal that excreted. He reduces this to the formula: $\frac{SI}{WI} + \frac{SP}{WP} = \frac{SE}{WE}$. In this formula S equals solids, W equals water, I equals ingested, P equals produced and E equals excreted. Thus, the solids and water ingested either orally or parenterally plus the solids and water produced by the metabolic process must equal the solids and water excreted. In Phase I there is an upset of this ratio because of decreased intake, increased metabolic activity due to fever, and decreased water output due to antidiuresis and the antidiuretic hormone. To be corrected, this ratio requires an increase in solid intake. Schlegel proposes intravenous administration of a solid, 4% urea, administered in 5% glucose, to solve this problem.

Phase II: Corticoid withdrawal phase: In this phase there is reversal of the changes effected during the corticoid phase, hence its name. Ushering in this phase is the return of gastrointestinal motility with passing of flatus. Local wound healing occurs with the elaboration of a mucopolysaccharide containing ground substance. Two other important components of the ground substance are hexosamine and chrondroitin sulfate. Tensile strength of the wound seems to parallel the content of 5 hydroxy proline. Here, even with exogenous proteins available, sulfur containing amino acids are still short in over-all body economy, due to their utilization in wound healing.

In the extracellular fluid, it is interesting to note that although the sodium and chloride are retained, the total serum concentration falls due to total body water increase. As this phase is ushered out there is a sodium diuresis and potassium retention. There is a reversal of the negative nitrogen balance, total body water decreases and body fat continues to be metabolized for energy.

Treatment during the state of corticoid withdrawal, potassium retention and sodium loss is directed toward early establishment of complete oral intake. During this stage the gastrointestinal tract becomes active and oral intake can begin. Parenteral administration should be decreased as oral feedings are increased. Wound healing is progressing normally but there is still a relative shortage of sulfur containing amino acids. If the kidneys are functioning normally, electrolyte balance can be controlled by oral administration and renal selective resorption and excretion. During this stage early ambulation will prevent circulatory stasis, thrombosis, edema and phlebitis.

In Phase I and II, vitamins are essential, especially the non-stored vitamins such as the vitamin B complex and those rapidly utilized such as vitamin C. The latter is utilized in wound healing, synthesis of adrenal steroids, and many of the body's oxidation and reduction enzyme systems.

Phase III. Spontaneous Anabolic Phase: In this phase protein stores and enzymes are replenished. The potassium to nitrogen excretion ratio is less than 2.1:1. Water and potassium enter the cell prior to matrix formation. Potassium is also needed intracellularly for glycogen storage and a relative potassium deficiency ensues. With anabolism, strength, interest and weight gain are seen in the patient. Wound healing continues, especially in terms of strength, and collagenous scar tissue is elaborated. The nitrogen balance is positive.

Treatment is directed toward the spontaneous anabolism. In this phase, the body requires a high protein, high carbohydrate diet with a high nitrogen to calorie ratio. During anabolic buildup, hypopotassemia may occur because potassium now enters the cell more rapidly than matrix is reformed. This can be corrected by adequate oral intake. An abundance of vitamins is needed. As in the first two phases, the patient gains strength and should attain complete ambulation; the wound should be protected from

great stress until tensile strength is adequate. As oral intake is possible now, parenteral therapy should be stopped.

Phase IV: Fat Gain: In this phase there is a return to a zero metabolic balance. The individual begins to replace the fat utilized in the previous phases. Sexual libido and potency return. Ultimate survival of the species is dependent on this final event.

Treatment must be prolonged as the fat gain phase may last for weeks or months. During this time the metabolic balance is restored and the fat depot is regained. In addition there is return of sexual function, menstruation in the female and potency in the male. Treatment is directed toward diet, exercise, and total rehabilitation.

Summary

Many investigators, including Bernard, Cannon, Seyle, Ramey, Moore and recently Dunphy and Edwards, have demonstrated a metabolic and systemic response to trauma carried down even to the cellular level. Many of the generally accepted hormonal effects and their interrelationship are diagramed and discussed. The four phases of post traumatic metabolism as described by Moore are given, as well as changes occurring throughout these phases, and some current thoughts about general treatment.

Conclusion

The metabolic changes occurring in the body following trauma are related to a systemic neuroendocrine response, and these responses are predictable. With knowledge of these, the predictable responses, reasoning can lead to more successful treatment of the surgical patient.

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MEDICAL CENTER

Message from the Dean

Children's Memorial Hospital as an integral unit of the University of Oklahoma Medical Center has three major functions—teaching, patient care and research.

The hospital is staffed to describe and demonstrate the best available methods of care for sick and handicapped infants and children. Achievement of this goal requires the ample and balanced supply of patient material provided by the State Crippled Children's program and by referring physicians throughout the state and area.

The preferred types of cases are diagnostic problems, medical and surgical problems which cannot be properly handled in the available community hospital, and patients who, in the opinion of the referring physician, present good teaching possibilities.

In an attempt to serve the physicians of Oklahoma as a referral and teaching center, Children's Memorial Hospital has certain special facilities:

In the Department of Pediatrics are specialists in pediatric cardiology, pediatric neurology, infectious disease, virology, metabolic and endocrine disorders, the handicapped child, speech and hearing disorders, and others. A number of practicing pediatricians in Oklahoma City and in surrounding communities participate in the teaching program.

The General Pediatric Outpatient Clinic is scheduled daily except Saturday and Sunday. The following special clinics: Child Guidance, Pediatric Neurology, Convulsive Disorders, Pediatric Allergy, Rheumatic Fever, Well Baby Clinic, Cardiac, Metabolic, Endocrine and Arthritis, are held regularly at less frequent intervals. The Infectious Disease Division has microbiological laboratories and medical and nursing personnel trained to care for patients with poliomyelitis, meningitis and other infectious diseases.

A Child Study Center provides comprehensive care for children with mental retardation, behavior disorders and other chronic or multiple handicaps. There are also the Premature Nursery, the Cardiovascular Unit for evaluation of children with various types of heart diseases, and a program for chemotherapeutic and surgical treatment of neoplastic disease.

The Orthopedic Service, which has been in successful operation for many years by the Department of Orthopedic and Fracture Surgery, conducts general and special clinics each week for children. Special services include such clinics as Scoliosis, Hand, Amputee and Cerebral Palsy.

Plastic Surgery, E.N.T., and Eye Clinics are held regularly, coordinated with inpatient care. Dental and orthodontia services are now available. A new multidiscipline Cleft Palate Conference is functioning as a rehabilitation consultation clinic.

A Pediatric Surgery Service has been developed as the key step in establishing a center for the study and care of surgical problems. Concentration of efforts in the new-born period is proving effective in the surgical group of congenital anomalies once considered hopeless. Thoracic and pediatric surgical clinics are held regularly. Since the state's first successful open heart operation at Children's Memorial Hospital in January, 1959, twenty-four of the 26 procedures in children under 15 years have been successful.

Children's Memorial Hospital is a general hospital for acute cases only. It cannot accommodate the long term chronically ill or patients requiring custodial care. There are no inpatient facilities for the severely emotionally disturbed child.

Mark R Everett

Griseofulvin, a new antibiotic effective against many fungi when administered orally, has received much recent attention. In this article the usefulness and dosage as well as limitations of this antibiotic are discussed.

Griseofulvin Therapy of Superficial Cutaneous Mycoses*

MARK ALLEN EVERETT, M.D.

HERMAN D. ALSTON, M.D.

In 1951, Mark Allen Everett, M.D., graduated from the University of Oklahoma School of Medicine where he is now an Instructor in Dermatology.

Certified by the American Board of Dermatology, Doctor Everett is a member of the American Academy of Dermatology and is now serving as President of the Oklahoma State Dermatological Society. In addition, he is Consultant in Dermatology to the United States Air Force.

Herman D. Alston, Jr., M.D., a graduate of the University of Arkansas School of Medicine in 1953, is a resident in dermatology at the University of Oklahoma School of Medicine.

ORAL THERAPY with griseofulvin for superficial fungous infection was first reported by Gentles[1] in August 1958. Following the preliminary report of the experimental use of this drug in humans by Blank[2] in December 1958 interest in the potentiality of griseofulvin became widespread. Subsequent studies have demonstrated the efficacy of this medication in the treatment of Trichophyton,[3], Microsporon,[4,5] and Epidermophyton infection of the superficial layers of the skin as well as in disease caused by Norcardia brasiliensis[6] and probably Sporotrichium schenkii.[7] Infections by other fungi, i.e., Norcardia, Blastomyces, Cryptococcus, and Histoplasma have not been influenced by this drug. Two common superficial fungi, Candida albicans and Malassezia furfur are not affected. Those fungi inhibited by griseofulvin reproduce only by hypheal growth so that inhibition by this antibiotic is brought about by the so-called "curling factor" as reported by Brian et al, in 1946.[8] Those fungi resistant to treatment with griseofulvin have an alternative method of reproduction, usually by budding.

In spite of the efficacy of griseofulvin in various fungous infections, duration and dosage of the drug is not firmly established. The following data is presented to assist in the formulation of dosage schedules.

Report of Cases

Nineteen patients with tinea capitis and seven with generalized tinea corporis due to T. rubrum were treated with griseofulvin** and patients were followed for a minimum of two months following cessation of therapy. Diagnoses were established by positive skin scrappings and the etiological agent was identified by culture. Three treatment schedules were followed: daily administration for 14 to 60 days; daily administration for 7-14 days followed by weekly administration; and weekly administration

*From the University of Oklahoma Medical Center, Department of Dermatology, John H. Lamb, M.D., Chairman.

**Griseofulvin (Fulvicin (R)) furnished by Schering Corp., Bloomfield, N.J.

TABLE I

TREATMENT OF TINEA CAPITIS WITH GRISEOFULVIN

Case No.	Patients	Causative Agent	Schedule	Duration	Result	1st Neg. KOH and culture at
1.	2½ w.f.	M. canis	combined	5 weeks	cured	21 days
2.	5 w.m.	M. canis	A) weekly	4 weeks	failure	—
			B) daily	3 weeks	cured	21 days
3.	5½ w.m.	M. canis	A) weekly	4 weeks	failure	—
			B) daily	5 weeks	cured	14 days
4.	5 w.m.	M. canis	combined	4 weeks	cured	21 days
5.	8 w.f.	M. canis	daily	3 weeks	cured	14 days
6.	4½ w.f.	M. canis	daily	6 weeks	cured	21 days
7.	10 w.m.	M. canis	A) weekly	4 weeks	failure	—
			B) daily	3 weeks	cured	14 days
8.	7 w.m.	M. canis	daily	5 weeks	cured	21 days
9.	5 w.m.	M. canis	combined	8 weeks	cured	21 days
10.	8 w.m.	M. canis	combined	6 weeks	cured	21 days
11.	3 w.f.	M. canis	combined	6 weeks	cured	21 days
12.	11 w.m.	T. tonsurans	daily	7 weeks	cured	28 days
13.	5 n.f.*	T. tonsurans	weekly	3 weeks	cured	28 days
14.	13 w.f.	T. tonsurans	daily	6 weeks	cured	14 days
15.	3 i.m.	T. tonsurans	daily	6 weeks	cured	21 days
16.	4 n.m.**	T. tonsurans	combined	3 weeks	failure?	—
17.	4 n.f.	T. tonsurans	daily	3 weeks	cured	14 days
18.	28 w.f.	T. schoenleini	daily	6 weeks	cured	21 days
19.	10 w.f.	T. schoenleini	daily	4 weeks	cured	21 days

*Medication discontinued on account of nausea but patient developed kerion
**Medication discontinued on account of nausea

only. Medication was continued for seven days beyond clinical and microscopic clearing of lesions. Patients were considered cured when they were clinically, microscopically and culturally free of infection two months after cessation of therapy. Daily administration consisted of 250 mg. q.i.d. to adults, t.i.d. to larger children (4-9 yrs.) and b.i.d. to small children. Single weekly doses ranged from two to three grams, according to weight. Topical treatment was limited to clipping of fluorescent hairs in all cases of tinea capitis.

Results

Results are tabulated in tables I and II. In *Microsporon canis* infections of the scalp, cures were obtained in all cases

TABLE II

TREATMENT OF TINEA CORPORIS WITH GRISEOFULVIN

Case No.	Patient	Disease	Schedule	Duration	Result	1st Neg. KOH and Culture
20.	59 w.f.	Extensive T. rubrum	combined	5 weeks	cured	28 days
21.	54 w.m.	Extensive T. rubrum	daily	6 weeks	cured	21 days
22.	48 w.m.	Extensive T. rubrum	weekly	6 weeks	failure	—
23.	18 w.m.	Moderate T. rubrum	daily	4 weeks	cured	21 days
24.	22 w.m.	Moderate T. rubrum	combined	4 weeks	cured	21 days
25.	32 w.m.	Extensive T. rubrum	daily	9 weeks	cured	42 days
26.	27 w.m.	Moderate T. rubrum	daily	4 weeks	cured	14 days

in which daily therapy was administered for fourteen days and beyond, the majority of patients receiving either weekly therapy for 3-6 weeks after fourteen days, or continuous therapy for 21-42 days. Those patients with only weekly therapy or with continuous therapy shorter than ten days failed to respond. Patients with *Trichophyton tonsurans* infection were clear after six weeks of daily therapy. Two patients with Trichophyton schoenleini infection were clear after 28 days of continuous therapy. In extensive *Trichophyton rubrum* infections of the skin, without nail involvement, no cures were obtained with only weekly therapy, while clearing occurred after 14 days continuous therapy followed by weekly therapy for four weeks or after 28-42 days of continuous therapy.

Side Effects

Three patients treated in this series had ill effects from griseofulvin. Two had nausea, vomiting, and headache. One developed fever and a maculopapular eruption. The drugs were discontinued in all cases.

Conclusions

It is felt that while this series is limited in extent the control of dosage and extent of follow-up permits a tentative conclusion regarding the duration of therapy necessary to produce a cure in a high percent of patients treated. In Microsporon infections of the scalp it is suggested that 0.5 to 1.0 gm griseofulvin daily (according to weight) for 28 days should produce a cure, provided that the infected hairs are clipped off or removed as they grow out. For the less common scalp involvement due to T. tonsurans or T. schoenleini six weeks of daily therapy is advised. For extensive T. rubrum infections of the skin, four to six weeks of daily therapy is advised, while in more limited involvement three weeks of daily therapy is probably adequate. Although not reported in this series, those cases where nails are involved with T. rubrum, a much longer course of therapy (four to eight months) is needed. Results are felt to be more consistent with daily as opposed to intermittent therapy.

Summary

Twenty-five patients with chronic fungous infection of the scalp and body were treated with griseofulvin. From the results a tentative plan of therapy is suggested. The need for microscopic confirmation of the diagnosis in each case is emphasized by the findings.

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The clinical aspects, pathologic physiology, differential diagnosis and surgical treatment of organic hyperinsulinism are discussed. Two illustrative case histories are presented.

HYPERINSULINISM

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ORGANIC HYPERINSULINISM is a symptom complex resulting from an excess of circulating insulin. An exogenous source may be responsible: insulin overdosage in a diabetic patient or surreptitious self administration of insulin. On the other hand, the excess insulin may be produced by a functioning tumor of the pancreatic islets of Langerhans. This entity is the condition under discussion.

The first successful removal of such a tumor was achieved by Graham in 1929.[1] In 1950 Howard et al[2] were able to collect from the world literature a total of 398 cases of which 224 had been operated. Virtually all of the signs and symptoms of organic hyperinsulinism result from a lowered fasting blood sugar. However, hypoglycemia should not be considered as being pathognomonic of hyperinsulinism for other disease entities involving organs normally participating in glucose metabolism may produce a low fasting blood sugar. A knowledge and understanding of this is essential for the differential diagnosis of the condition under discussion. The various mechanisms by which hypoglycemia may be produced are readily understood when examined in the light of some of the physiologic processes involved in the metabolism of glucose.

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Physiological Aspects

Glucose is the chief fuel of muscular exercise and if unavailable in the diet must come from conversion of protein and fat molecules. Whereas carbohydrate is stored in the liver and tissues in the form of glycogen, the central nervous system has little glycogen. Since only glucose can be oxidized by brain tissue,[3] the central nervous system is absolutely dependent upon a minute by minute supply of glucose from the blood. Without any available glycogen reserve the nerve cell very rapidly loses its functional integrity in the face of hypoglycemia. This explains the rapid development of hypoglycemic symptoms when the blood sugar is

lowered. The mechanism of these symptoms is a depression of oxidative processes in the brain resulting in cellular damage similar to that occurring in anoxia from other causes.[4,5] According to Moersch and Kernohan[4] the most important damages noted in post mortem examinations following severe insulin reactions are nerve cell degeneration and multiple petechial hemorrhages. These changes are very similar to those seen in the brain of patients dying of cerebral anoxia.

Several regulatory agencies operate in maintaining a relatively constant level of blood sugar. One of these is the capacity of the liver to store glucose in the form of glycogen and in turn release glucose when needed. The liver also maintains the carbohydrate reserves by converting protein and fat molecules into carbohydrate.

Insulin from the pancreatic islets is the only hormone concerned with glucose metabolism that tends to lower the concentration of blood sugar. The mechanism by which this is accomplished is an increase in the peripheral utilization of glucose by the tissues. It appears that insulin accomplishes this by increasing the permeability of muscle cells to glucose.[6]

There is between the metabolism of carbohydrate and the adenohypophysis a relationship that has been evident clinically for some time. Although the incidence of diabetes in the general population is about 0.5%, the disease occurs in acromegalics in about 25-40% of cases.[3] On the other hand, hypoglycemia is often a feature of hypopituitarism. The mechanism underlying the influence of the anterior lobe of the pituitary gland on the metabolism of carbohydrate is not completely elucidated. It appears that growth hormone may inhibit carbohydrate utilization by decreasing lipogenesis from carbohydrate and acetate.[7] Furthermore, anterior pituitary secretion of ACTH also tends indirectly to elevate the concentration of blood sugar by stimulating the secretion of adrenocortical steroids. These hormones are effective in promoting glyconeogenesis. This is accomplished by increasing the rate of protein catabolism and rendering more intermediary metabolites available for conversion into glycogen by the liver.[8] In practice, patients with hypopituitarism or primary hypoadrenocorticism may have a low fasting blood sugar. Also, it is well to remember that patients with pituitary insufficiency are extremely sensitive to insulin so that a small amount of insulin which would produce no noticeable effect on a normal individual may in a patient with pituitary insufficiency cause prolonged and even fatal hypoglycemia.[9]

The adrenal medulla also contributes to blood sugar regulation by discharging larger amounts of epinephrine when the blood sugar falls below 50 mg%. Epinephrine mobilizes the preformed stores of glycogen in the liver and muscle and rapidly elevates the blood sugar. Many of the symptoms of hypoglycemic crisis such as tremor, irritability and sweating are merely the systemic manifestations of epinephrine.

Clinical Aspects

The signs and symptoms of hyperinsulinism are directly related to the hypoglycemic state. Moersch and Kernohan[4] described three stages of symptoms. The first, one of retardation, may last for ten to thirty minutes during which the patient appears mentally and physically retarded. Transitory diplopia, tremor, irritability, restlessness and confusion may be present. Drowsiness may be the only symptom. If the hypoglycemia is not averted, a stage of excitement with pallor, marked perspiration and bizarre psychic behavior ensues to be followed in turn by a phase of depression with coma during which one may find signs and symptoms of organic brain disease such as positive Babinski reflexes, motor palsy, hemiparesis, etc. These signs usually disappear after recovery from the hypoglycemia. Hypoglycemic attacks occur most frequently in the late afternoon or before breakfast. Often the attacks are precipitated by physical exertion. Patients with hyperinsulinism soon learn that they are able to alleviate or even prevent hypoglycemic attacks by eating large amounts of carbohydrate. Some patients even go to the extreme of setting their alarm clock to awaken them in the early morning hours so that they may eat and

avoid morning symptoms. As a consequence of this increased dietary intake, many patients with hyperinsulinism are obese. Whipple[10] found that obesity was present in 24 out of 31 patients with organic hyperinsulinism.

The preceding statements regarding the symptomatology of organic hyperinsulinism are summarized in the classical symptomatic triad known as Whipple's triad: Attacks of hypoglycemic symptoms associated with fasting or exercise, . . . accompanied by a blood sugar lower than 50 mg%, . . . and relieved by the administration of glucose.

The most important aid to the diagnosis is an awareness on the part of the physician that hyperinsulinism may be responsible for such vague and unspecific symptoms as listed in table 1. Unless the examiner entertains this possibility, he may omit to question the patient regarding the relationship of the attacks to fasting or exercise. Such a line of questioning is the key to the clinical diagnosis of hyperinsulinism. If this appears to be stressing the obvious, one need only recall that the initial diagnosis was erroneous in 46 out of 91 cases of hyperinsulinism recently reviewed by Breidahl.[11] In view of the nature of the presenting symptoms it is not surprising that such diagnoses as epilepsy, psychosis or inebriation are considered. It is unfortunately true that when a patient has such diagnoses on his record the chances of his hyperinsulinism being recognized at a later date diminish. Early diagnosis of this condition is of the utmost importance because repeated hypoglycemic attacks may lead to irreversible brain damage.

TABLE 1.

Signs and Symptoms Exhibited by 193 Patients with Insulinomas*

Sign or Symptom	Per cent
Loss of consciousness	58
Confusional state	54
Weakness and fatigue	41
Deep coma	40
Sweating	36
Drowsiness and stupor	35
Lightheadedness	30
Visual Disturbances	30
Amnesia	28
Clonic convulsions	24
Noisy behaviour	20
Headache	20
Tremor	18
Hunger	14
Positive Babinski sign	13
Paresthesias	13
Irritability	11
Transient hemiplegia	10
Abdominal pain	8
Palpitation	3

*Reprinted from Crain, E. L., Jr., and Thorn, G. W.: Functioning pancreatic islet cell adenomas; a review of the literature and presentation of two new different tests. Medicine 28: 427-447, 1949.

Of course the alternate pitfall is a hasty diagnosis. Reasons for caution in this regard are obvious if one remembers that the only form of treatment is surgical, often of a radical nature.

Inasmuch as the fundamental objective alteration in this condition is a low fasting blood sugar, it is imperative that several blood sugar determinations be done. The diagnosis of organic hyperinsulinism is strengthened in proportion to the number of blood sugar determinations lower than 50 mg%. Occasionally the fasting blood sugar may be only equivocally lowered or even normal. This necessitates provoking a hypoglycemic attack by fasting or exercise. All food except for unsweetened tea or coffee and water is withheld from the patient until hypoglycemic symptoms appear. Rarely does one have to wait for more than a few hours. However, the fast may have to be prolonged for forty-eight to seventy-two hours before symptoms appear. As soon as the latter become evident, blood is withdrawn for blood sugar analysis and glucose is administered parenterally. If the symptoms are on the basis of hyperinsulinism, the administration of glucose should bring about their immediate relief. A hypoglycemic attack may be provoked by carefully supervised exercise on the ward. The test should be terminated as described above as soon as the first symptoms appear. In general, it is agreed that the glucose tolerance test is of little value in hyperinsulinism.[12] Results of the test may vary from flat to diabetic types of curves.

The following two cases are presented as an illustration of the relative urgency for definitive diagnosis in hyperinsulinism.

Case 1: The patient, a 39 year old white male mechanic, was admitted to the V.A. Hospital, Oklahoma City for the first time in July of 1954. The following history was obtained from his wife. He had been well until September 1953 when he began to experience recurrent attacks of unconsciousness. The first attack occurred in September 1953. While at work and just before eating his noon meal, he suddenly slumped over. He regained consciousness one hour later. A diagnosis of heat stroke was made. Two months later, while working around the house, he exhibited irrational behaviour for several minutes and then slumped over. He remained unconscious for five hours. His wife noticed some jerking of his extremities. Shortly thereafter he was hospitalized elsewhere for two weeks. No diagnosis was made. Upon returning home he had another episode during which his wife noticed clonic convulsions over the entire body. He was admitted in a coma to another hospital and remained comatose for 18 hours. During that hospitalization a diagnosis of left cerebral atrophy was made. No blood sugar determinations were done. All other laboratory studies, serology, hemogram and urinalysis were normal. The skull films were normal and the EEG pattern was not altered. He was discharged under observation for convulsive disorder. In July 1954 another attack occurred during which he remained comatose for about 30 hours before being brought to this hospital. His wife stated that since the last hospitalization he had been unable to hold a job on account of chronic fatigue.

An EEG was done and showed a slow rhythm of low amplitude over the left side suggesting a functional illness. Routine laboratory determinations were within normal limits. Blood sugar determinations were not done. A diagnosis of conversion reaction manifested by periods of amnesia and somnolence was made.

He was readmitted to this hospital in August of the same year after having been comatose for approximately 72 hours. He was first admitted to the medical service. Because of the previous diagnosis of conversion reaction he was transferred to the psychiatry service. While on this service, the patient developed very irrational behaviour and seemed out of contact with his environment. He became more and more lethargic until he no longer responded to stimuli. He improved after parenteral and tube feeding. Except for a transitory positive Babinski reflex, the physical examination was negative. Deep tendon reflexes were active and cranial nerves were intact. No sensory impairment was noted. Spinal fluid examinations were normal. A pneumoencephalogram was interpreted as showing minimal to moderate cerebral atrophy on the left side. No blood sugar determinations were done. A diagnosis of chronic brain syndrome of unknown cause and left cerebral cortical atrophy was made.

After discharge the patient had recurrent attacks of unconsciousness lasting from fifteen minutes to four hours. In November 1954 he was hospitalized elsewhere for eight days without a definitive diagnosis being made. His last admission to this hospital was in December 1954. Five days prior to admission the patient had another attack of unconsciousness which lasted for five days during which he was either completely unconscious or in a semiconscious state. Clonic convulsions of all extremities occurred several times during this period. On the day of admission the patient was placed on the psychiatric service due to the previous diagnosis of chronic brain syndrome. The following day the patient was seen by a neurosurgical consultant and a diagnosis of possible organic brain disease was made and the patient was transferred to the neurosurgical service. A routine blood sugar determination was found to be 29 mg%. It was repeated and a level of 33 mg% was found. Following this report, the patient was given 70 ml of 50% glucose intravenously with immediate return of consciousness. A diagnosis of islet cell tumor of the pancreas was made. The patient was prepared for operation and three weeks after admission an exploratory laparotomy was carried out. A 2 cm by 1 cm round mass was palpated

anteriorly in the head of the pancreas just above the uncinate process. The mass was removed by wedge resection. On histologic examination the lesion was found to be a benign beta-cell adenoma. There were no surgical complications except for some drainage of pancreatic secretions which ceased completely after five weeks. During the remainder of his hospitalization the patient showed a gradual return of memory and a loss of irritability. His blood sugar came back to a normal level. Psychological examination demonstrated a general dysfunction in the higher, more complex mental processes. It was estimated that his intelligence had at one time been above average. Although it was considered likely that he would regain more intellectual efficiency, it was thought that he would continue to show organic impairment. The patient was discharged two months after the date of his last admission.

A social service follow-up six months later revealed that, although the patient had not had any more episodes of unconsciousness, his behaviour was still abnormal. He had temper tantrums at work and at home and remained hostile and belligerent. When agitated or excited he reacted in a hostile, explosive, impulsive manner to any demands placed upon him by his family or friends.

In spite of successful surgical removal of the functioning islet-cell adenoma the patient had a poor result because of irreversible organic brain damage brought on by repeated severe hypoglycemic attacks. Unfortunately, throughout the patient's successive hospitalizations a psychiatric diagnosis served as a "mental block."

Case 2: The patient, a 57 year old unemployed male, was admitted to the V.A. Hospital, Oklahoma City in June 1958. He gave a history of fainting, a week before admission, while getting out of bed in the morning. This happened before breakfast. He remembered waking up in the hospital. Apparently, he remained unconscious for five hours. No clonic convulsions were noticed during this time. He was discharged from the other hospital without any definitive diagnosis being made. The patient denied any previous episodes of unconsciousness, syncope or convulsions. However, during the past year he had experienced episodes of blurred vision with diplopia lasting from one to thirty minutes. These attacks occurred every two weeks and subsided spontaneously. When they occurred while he was driving, he noticed that the center line of the road seemed to diverge to one or the other side of the road. The patient noticed that the attacks were worse or were more prone to occur when he missed his noon meal or had been working hard. On further questioning, he said that his memory was not as good as it had been. He also admitted that he was more irritable and nervous than he used to be and had to quit his job in a furniture factory because of differences with his employer. He had not noticed any craving for sweets but had gained approximately 20 lbs. during the past year.

The past history revealed that the patient had been discharged from the army in 1931 with the diagnosis of rheumatic heart disease.

On physical examination the patient was found to be a slightly obese, healthy looking male. Except for a grade 2 apical systolic murmur, the physical examination was negative. There were no positive neurological findings. However, it was apparent that the patient was rather vague in his answers to questions and seemed unable to focus his attention on what was going on around him.

Two days after admission, a fasting blood sugar was 52 mg%. Subsequently, 12 fasting blood sugar determinations were done. Of these, nine were below 40 mg%; the lowest value was 26 mg%. While being fasted on the ward for the latter blood sugar determination, the patient became suddenly unconscious. Blood was withdrawn for sugar analysis and an intravenous drip of glucose in water was started. Immediate remission occurred. Three weeks after admission the patient was explored through a bilateral subcostal incision. A 1 cm by 1 cm nodule was palpated in the tail of the pancreas, approximately 4 cm proximal to the tip of the gland. A distal pancreatectomy and splenectomy were performed. Six hours after operation the blood sugar was 190 mg% and glucosuria was present. Insulin

was required for forty-eight hours. The post operative course was uncomplicated. On histologic examination the lesion was found to be a benign beta-cell adenoma of the pancreas.

The patient has been unsymptomatic since the operation. His memory has returned completely and he has had no further episodes of weakness or of blurred vision.

It is obvious that the only difference between the two cases was the prompt diagnosis made in the latter case by the Medical Service. This is what leads to a full recovery after operation.

Differential Diagnosis

A) Endogenous hyperinsulinism due to a functioning pancreatic tumor must be differentiated from unrecognized exogenous hyperinsulinism produced by the surreptitious self administration of insulin.[13] Obviously such patients have signs and symptoms that are identical to those present in the condition under discussion. Often these individuals submit to repeated surgical procedures which compound the difficulty of making the proper diagnosis. The only clue is the discovery of insulin or syringes in the patient's room. One should think seriously of this possibility when dealing with patients such as nurses or relatives of diabetics who have had some experience with insulin. The recognition of chronic insulin overdosage in a diabetic should not be a problem.

B) As was mentioned above, the physiology of glucose metabolism is such that disease processes involving glands or organs other than the pancreas may produce a low fasting blood sugar.

Severe liver disease with failure of gluconeogenesis is an occasional cause of spontaneous hypoglycemia. Mann and McGath[14] originally observed that one of the most striking effects of hepatectomy in the dog was a profound depression of blood sugar resulting in hypoglycemic coma and death. In clinical practice, the recognition of hepatogenic hypoglycemia should not be difficult. In patients with massive liver metastases, with far advanced cirrhosis, with terminal infectious hepatitis, the hypoglycemic manifestations are usually overshadowed by other obvious evidence of severe liver disease.

Patients with pituitary insufficiency are prone to have a low fasting blood sugar. The other signs of the syndrome should enable one to rule out hyperinsulinism. These patients are extremely sensitive to insulin and if an insulin sensitivity test is contemplated only very small amounts of insulin should be used.

The Addisonian patient often has a low blood sugar. The pigmentation, low blood pressure and characteristic electrolyte abnormalities should point to the correct diagnosis. Needless to say, the consequences of surgical exploration in search of a non existent insulinoma in a patient with unrecognized Addison's disease could be catastrophic.

Finally, there is an ill defined group of patients suffering from a condition known as functional hypoglycemia. The symptoms are similar to those of organic hyperinsulinism. However, they are generally of a mild degree and consist mainly of attacks of weakness and dizziness. The symptoms may last for years and do not increase in severity from one year to the next in contrast to those of hyperinsulinism in which the symptoms progress steadily. As has been stressed by Keating and Wilder,[15] the symptoms of functional hypoglycemia occur several hours after meals whereas those of hyperinsulinism are brought on by fasting. Furthermore the blood sugar usually remains over 50 mg% even during an attack. A glucose tolerance test is useful in that it may reproduce the symptoms and reveal a sharp dip in blood sugar associated with the symptoms during the second to fourth hour of the test. The symptoms improve if the patient is placed on a high protein, low carbohydrate diet. Characteristically, patients with functional hypoglycemia are emotionally labile. Hypoglycemia of a functional nature is also an occasional complication of gastric operation. Patients in whom a gastric resection or gastro-enterostomy have been performed may develop hypoglycemic symptoms several hours after a meal. All patients who have had this type of operation have a glucose tolerance test characterized by an initial

hyperglycemia to diabetic levels with a delayed and sharp drop in the concentration of blood sugar. However, in only a small percentage of patients will this delayed hypoglycemia be symptomatic. The symptoms should be distinguished from those of the dumping syndrome which are manifest immediately after a meal.

Although islet cell adenomas have been removed from infants, it remains true that hypoglycemic states in infants and children are less likely to be due to a functioning pancreatic tumor that they are in the adult. Idiopathic hypoglycemia of children or McQuarrie's disease is of hereditary origin and thought to be due to an absence of alpha cells in the islets of Langerhans.[16] Since these cells appear to produce a hyperglycemic-glycogenolytic factor (Glucagon), their absence may be responsible for the hypoglycemia. ACTH was found by McQuarrie to be extremely effective in controlling hypoglycemic symptoms in these children as long as it was administered.

Treatment

The treatment of hyperinsulinism is surgical removal of the hyperfunctioning islet cell tumor of the pancreas. In our opinion, the fundamental prerequisite to surgical treatment of this lesion is an absolute confidence on the part of the operating surgeon in the clinical diagnosis prior to submitting the patient to laparotomy. The surgeon should participate in the clinical work up of the patient in order that he may open the abdomen not for the purpose of finding out if a tumor is present but of removing the tumor that he knows is present. Frequently the offending lesion is far from obvious and its discovery and removal may require major surgical maneuvers. It is unfortunately true that these patients often undergo several half hearted surgical explorations before the lesion is finally found and removed.

Glucose, in the form of 10% glucose in water should be given before and during the operation. The prolonged preoperative fast and the increased liberation of insulin from the tumor incident to its manipulation may lead to a dangerously low blood sugar level.

The abdomen is opened through a bilateral subcostal incision with division of both recti. The gastro-colic ligament is incised widely. Attention is directed first to the body and tail of the pancreas since the majority of insulinomas are in this location. In the absence of a palpable tumor on the surface of the gland, the peritoneum along the inferior margin of the pancreas is incised and the body and tail mobilized with gentle finger dissection. This maneuver enables one to palpate the tail and body of the pancreas between the thumb and fingers up to the level of the mesenteric vessels. An adenoma, when present, can be felt as a rounded mass of a consistency slightly firmer than that of the pancreas. In the event of negative exploration of the distal portion of the pancreas, the head of the gland and surrounding duodenum are mobilized by division of the peritoneum along the lateral border of the second portion of the duodenum (Kocher's maneuver). With finger dissection the pancreatic head is separated from the posterior planes to the level of the mesenteric vessels medially. The gland can then be palpated between the thumb and fingers. Care must be taken not to overlook the uncinate process during this part of the exploration. If nothing is found in the head of the pancreas, the search is directed towards the common sites of pancreatic heterotopia where aberrant pancreatic insulinomas are occasionally found. These are the duodenal wall, the wall of the gastric antrum, the gastro-splenic omentum, the hilum of the spleen and the retroperitoneal tissues between the head of the pancreas and the liver. Howard et al[2] from a collected series of 398 cases reported a 2% incidence of ectopic islet cell tumors.

In approximately 75% of the cases the tumor is located in the body and tail of the gland.[2] In approximately 10% of cases the lesion is a functioning islet cell carcinoma.[11] Multiple tumors have been reported in from[11] 2% to 10% of cases.[2] In relation to the multiplicity of islet cell tumors, it seems worthwhile to mention the existence of multiple endocrine tumors of which at least 22 cases have been reported in the world literature.[17] These patients had adenomas of the pituitary body, the parathyroid glands and the pancreas or had tumors in some combina-

tion of two of these glands. Multiple pancreatic islet cell adenomas were present in eight such cases reported from the Mayo Clinic.[18] Because of the high incidence of multiple tumors it is imperative that one should proceed with careful exploration of the entire gland even when a tumor has been found immediately.

The size of islet cell adenomas is rarely greater than 2 cm in diameter.[11] Unfortunately for the operating surgeon small tumors are not uncommon. At least one instance of an adenoma 2 mm in diameter has been reported.[19] The tumor was removed during a blind subtotal pancreatectomy and was not found during a routine pathologic examination of the specimen. Only several years later was the lesion found during serial sectioning of the specimen. In the meantime the patients symptoms had been cured.

In some cases no tumor can be found after a careful exploration. The incidence of negative explorations varies from 16% to 35%.[20] This incidence can be kept at a minimum by following rigourous criteria for the preoperative diagnosis of hyperinsulinism. Diffuse hyperplasia of the islets of Langerhans has been suggested as a possible cause of hyperinsulinism particularly in the cases where there has been a negative exploration.[21] However, there is no evidence to suggest that such an entity exists.[22]

The management of the lesion is directed by the findings. In view of the relatively high incidence of malignant changes in islet cell tumors, many authors agree that a lesion in the tail of the gland is best treated by a conservative distal pancreatectomy, thus encompassing the lesion rather than enucleating it.[20] But when an adenoma is situated in the head of the pancreas, the advantages of this "tumor approach" are outweighed by the high mortality (as high or higher than the incidence of malignant insulinomas) and morbidity of pancreaticoduodenal resection. In such a situation, simple enucleation or wedge resection is the procedure of choice. Of course, when one is faced with a frank islet cell carcinoma with metastases, it is justifiable to remove as much of the primary as possible in an effort to palliate otherwise uncontrollable hypoglycemic symptoms. When a thorough exploration fails to reveal a palpable lesion, one surmises that the tumor is too small to be detected by palpation (assuming that preoperative criteria for hyperinsulinism have been rigorously followed). Because of the high probability of the lesion being in the tail or body of the pancreas, the portion of the gland situated to the left of the mesenteric vessels should be removed.[11] Frequently, following such a "blind" resection, one is gratified by finding a minute adenoma in the surgical specimen and by the subsequent disappearance of the patient's symptoms. If the latter persist after a blind distal pancreatectomy, there is no alternative other than to reexplore the patient and in the event of another negative exploration, to resort to removal of the remainder of the gland. Fortunately, such an eventuality presents itself only infrequently. However, it should be emphasized that the management of diabetes following total pancreatectomy is less of a problem than the management of severe hyperinsulinism.[23]

Postoperatively all of the patients have a transitory diabetes lasting for twenty-four to seventy-two hours. This phenomenon, i.e., the depression of the function specific to the tumor, is common to all endocrine tumors following their removal. Sufficient insulin to cover the glucose in the intravenous infusion should be given. Dehydration should be prevented for these patients may have a greater urinary output during the early postoperative period than the average surgical patient.

Results of Treatment

In the majority of instances, the results of surgical treatment are excellent. This is particularly true of those cases in which the lesion is found at exploration, is single and benign, and can be removed by a partial pancreatectomy. Good late results under such circumstances can be expected in about 90% of cases.[11] When a blind distal pancreatectomy is performed because of failure to find a lesion, approximately 60% of the patients are cured of their symptoms.[24] The results

of surgical treatment of islet cell carcinomas are uniformly poor.[20]

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ABSTRACTS

Canine Atherogenesis Following I^{131} Administration and Cholesterol Feeding

I. G. GONZALEZ,* L. N. NORCIA,** M. R. SHETLAR,*** C. W. ROBINSON,**** L. L. CONRAD***** and R. H. FURMAN.******

American Journal of Physiology, 197: 413-422, 1959

Atherogenesis in I^{131}-treated cholesterol-fed dogs was evaluated utilizing tissue lipid and polysaccharide, serum lipid and lipoprotein analyses. Coronary artery lesions antedated aortic lesions and both were characterized first by ground substance change without lipid accumulation. Lipids other than cholesterol then appeared, associated with varying degrees of cellular proliferation, subintimal hemorrhage and involvement of vasa vasorum and media. Marked cholesterol but not phospholipid accumulation characterized late lesions. Metachromasia was noteworthy only in fibrotic lesions. Aortic tissue hexose and hexosamine values usually were normal. Liver analyses revealed increased glycogen, especially in animals with marked reduction in high density serum lipoproteins. These animals also had highest concentrations of serum cholesterol, lipid P and lower density lipoproteins, and the most severe atheromata. Serum C/P ratios increased with increasing cholesterol levels. At serum cholesterol levels above 1000 mg %, the increments in C/P ratios were relatively smaller. Hypercholesterolemia in excess of 450% for one year appears requisite to atherogenesis in the I^{131}-treated cholesterol-fed dog, while comparable hypercholesterolemia in I^{131}-treated dogs not fed cholesterol does not lead to atheroma formation.

*Department of Pathology, University of Alabama School of Medicine.

**Assistant Professor of Research Biochemistry.

***Associate Professor of Biochemistry.

****Medical Resident.

*****Assistant Professor of Medicine.

******Associate Professor of Research Medicine.

Serum Lipid, Lipoprotein and Vascular Tissue Studies in Cholesterol-Fed Horse

L. N. NORCIA,* W. JOEL,** and R. H. FURMAN.***

Proceedings of the Society for Experimental Biology and Medicine, **100**: 759-761, 1959

A diet containing 1.7-2% cholesterol did not alter serum lipid or lipoprotein levels when fed to a gelded horse for three months. When cholesterol supplementation was discontinued, significant increases in serum total and free cholesterol, lipid phosphorus, C/P ratio and high density -$S_{1.21}$0-20 ("alpha") lipoproteins occurred which persisted until sacrifice three months later. Fecal lipid analyses indicated net cholesterol absorption in excess of 70%. Liver and aortic lipid analyses and histologic examination of myocardium, coronary arteries and aorta were not remarkable.

*Assistant Professor of Research Biochemistry.
**Professor of Pathology.
***Associate Professor of Research Medicine.

The Effect of Bilateral Pulmonary Resection of Total Oxygen Uptake and Pulmonary Hemodynamics inthe Dog

MICHAEL T. LATEGOLA,* WALTER MASSION,** and JOHN A. SCHILLING,*** Oklahoma City, Okla.

The Journal of Thoracic Surgery **37**, 606-610, 1959

The $V_{o2\ T}$ and Q-T are only slightly and insignificantly altered following 56.5 per cent bilateral resection of the whole lung. The PAP and R-T are increased following both resections but disproportionally increased following the second resection.

*Assistant Professor of Physiology.
**Research Associate in Anesthesiology.
***Professor of Surgery.

Quantitative Response of the Rat Ovary To Pituitary Gonadotropin as Modified By Estrogen

R. W. PAYNE* and R. H. RUNSER.**
Endocrinology, **65**: 383-388, 1959

A purified pituitary gonadotropin containing predominantly FSH activity was administered in graded doses to hypophysectomized and intact immature female rats, with and without concurrent administration of diethylstilbestrol in large doses.

A more striking and consistent ovarian weight increase to pituitary gonadotropin, brought about through stimulation of early ovarian follicle development, regulation of further follicular progression, and depression of interstitial tissue overgrowth was observed in the diethylstilbestrol-treated hypophysectomized animals.

*Associate Professor of Pharmacology and Instructor in Medicine.
**Intern, Mary Imogene Bassett Hospital, Cooperstown, New York.

Wound Healing: Glycoproteins of Wound Tissue I. Studies of Hexosamine, Hexose, and Uronic Acids Content

M. R. SHETLAR,* EUGENIA G. LACEFIELD,** BETTY N. WHITE,*** and JOHN A. SCHILLING.****

Proceedings of the Society for Experimental Biology and Medicine **100**: 501-503, 1959

A serial study of uronic acid, hexosamine, and hexose content of wound repair tissue was made. Uronic acid content was quite low in the seven-day sample, but increased until about 28 days. Hexosamine in considerable excess of uronic acid was found indicating presence of polysaccharides or glycoproteins which did not contain uronic acid. Sugars with electrophoretic mobility in borate buffer of galactose, mannose, and glucose were identified in hydrolysates of wound repair tissue.

*Associate Professor of Biochemistry, University of Oklahoma Medical School.
**Technician, Department of Biochemistry.
***Research Technician, Department of Surgery.
****Professor of Surgery

Poliomyelitis Problems in the Post-Vaccine Era

HARRIS D. RILEY, Jr.,* and RANDOLPH BATSON

Southern Medical Journal, **52**: 127-135, 1959

Physicians have been inclined to accept poliomyelitis vaccination as insurance against future problems with this disease. This has been a summary of the difficulties that will continue to exist in a population immunized largely with killed-virus vaccine. Suggestions for counteracting these problems have been made.

*Professor and Chairman, Department of Pediatrics.

In Vitro Activation by Tocopherol of the Synthesis of Ascorbic Acid by Liver Extracts Of Tocopherol-Deficient Rats

PAUL B. McCAY,* MARY P. CARPENTER,** ABBAS E. KITABCHI,*** and RANWEL CAPUTTO.****

Archives of Biochemistry and Biophysics, **82**: 472-473, 1959

*Instructor in Research Physiology.
**Biochemist, Oklahoma Medical Research Foundation
***Research Associate, Oklahoma Medical Research Foundation.
****Associate Professor of Research Biochemistry.

Non-Hypocholesterolaemic Action of Nicotine Acid in Dogs

L. N. NORCIA,* H. J. BROWN,** and R. H. FURMAN.***

Lancet **1**: 1255, 1959

*Assistant Professor of Research Biochemistry.
**Intern, Walter Reed Hospital, USAF.
***Associate Professor of Research Medicine.

Heatley Named Professor Emeritus

The Alumni Association of the University of Oklahoma School of Medicine honored John E. Heatley, M.D., Oklahoma City, as Professor Emeritus of the Year during the annual banquet meeting October 25 at the Skirvin Tower Hotel, Oklahoma City.

HEATLEY

John R. Taylor, M.D., Kingfisher, former president-elect, was installed as Association president, succeeding Carl H. Bailey, M.D., Stroud.

Doctor Heatley, a former chairman of the Department of Radiology, was appointed professor emeritus of diagnostic radiology in 1949. Ill health prevented his attending the program and James R. Reed, M.D., Oklahoma City, received the plaque for him.

A "Professor Emeritus" plaque also was presented Wann Langston, M.D., professor emeritus of medicine, who was cited in 1957. The practice of presenting plaques to the honorees was not started until last year and Doctor Langston was in Europe when the presentation to past honorees was made at the 1958 session.

Alumni voted to abolish the office of president-elect. They re-elected W. F. Lewis, M.D., Lawton, vice-president and Samuel T. Moore, M.D., Oklahoma City, secretary; and elected Wayne A. Starkey, M.D., Altus, treasurer.

Four new trustees also were named. They are Hillard E. Denyer, M.D., Bartlesville, representing District 7; O. L. Parsons, M.D., Lawton, District 23; Malcolm Mollison, M.D., Altus, District 24; and William T. Snoddy, M.D., Oklahoma City, District 28.

Five-year reunion classes, beginning with the Class of '09, were introduced. Sole member of the Class of 1909 attending was Charles R. Hulen, M.D., Ardmore. Joseph T. Phelps, M.D., El Reno, represented the Class of 1919.

Regents Appoint Worsham and Hudnut

Two appointments to the School of Medicine faculty were approved by University of Oklahoma Regents at their October meeting.

WORSHAM

Bertrand Ray Worsham, M.D., was named clinical assistant in psychiatry, neurology and behavioral sciences, and Herbert Beecher Hudnut, Jr., M.D., was appointed visiting lecturer in preventive medicine and public health.

Doctor Worsham received his M.D. degree in 1955 from the University of Arkansas School of Medicine. He interned at Hillcrest Medical Center, Tulsa, and took residency training at Menninger School of Psychiatry, Topeka, from 1956 to 1958 and at the OU Medical Center from 1958 to 1959.

Doctor Hudnut, a 1957 graduate of Harvard Medical School, interned in medicine and served a one year medical residency at Mary Imogene Bassett Hospital, Cooperstown, N.Y.

HUDNUT

He has participated with Doctor William Jaques, chairman of the Department of Pathology, in studies of the relation of atrial pathology to cardiac arrhythmias. Research also includes analyzing data from the Carter county cardiovascular screening program conducted in 1958.

THIRD OKLAHOMA COLLOQUY

on

ADVANCES IN MEDICINE

March 24, 25, 26, 1960

University of Oklahoma Medical Center

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Clinical reports on Dartal: 1. Edisen, C. B., and Samuels, A. S.: A.M.A. Arch. Neurol. & Psychiat. *80*:481 (Oct.) 1958. 2. Ferrand, P. T.: Minnesota Med. *41*:853 (Dec.) 1958. 3. Mathews, F. P.: Am. J. Psychiat. *114*:1034 (May) 1958.

SEARLE

We Wish You
a Merry Christmas
and a Happy New Year

The Agnes T. Bakers

Coming Meetings

UNIVERSITY OF OKLAHOMA MEDICAL CENTER
POSTGRADUATE PROGRAM*
Individual Postgraduate Courses

BASIC ELECTROCARDIOGRAPHY — February 29 through March 5.

OPTHALMOLOGY-OTOLARYNGOLOGY SYMPOSIUM—March 3 and 4.

OBSTETRICS-GYNECOLOGY SYMPOSIUM—March 5.

ADRENAL STEROIDS—March 24, 25, and 26. (Third Oklahoma Colloquy on advances in Medicine.)

ORTHOPEDIC SYMPOSIUM—May 13 and 14.

CARCINOMA OF THE SKIN—April 22 and 23. (Sixth Annual Combined Surgery, Radiology, Pathology Symposium.)

OKLAHOMA ASSSOCIATION OF HOUSE STAFF PHYSICIANS—May 6.

PEDIATRIC NEUROLOGY—May 12 and 13. This symposium to be held in conjunction with the Wednesday Short Course on May 11.

SERIAL POSTGRADUATE COURSE
Postgraduate Division
Oklahoma City, Oklahoma
1959-1960

Wednesday Short Courses
3:30 to 8:30 p.m.

Jan. 13	Carl Puckett Memorial Lecture and Infectious Diseases Symposium Guest Lecturer: William Tucker, M.D., Washington, D.C.
Feb. 10	Rehabilitation of the Cardiac Patient
Mar. 9	C. B. Taylor Lectureship and Urology Symposium—Guest Lecturer to be announced
Apr. 13	Anesthesia for the Part-time Anesthetist
May 11	Neurological Diseases in Childhood
June 8	Surgery

*Further information may be obtained from the Office of Postgraduate Education, University of Oklahoma School of Medicine, 801 N.E. 13th Street, Oklahoma City, Oklahoma.

OKLAHOMA CHAPTER
AMERICAN ACADEMY OF GENERAL PRACTICE
February 15-16, 1960 — **Tulsa Hotel**
Tulsa, Oklahoma

The 12th Annual Meeting of the Oklahoma Chapter of the American Academy of General Practice will be held at the Tulsa Hotel, February 15-16, 1960, in Tulsa.

EIGHTH POSTGRADUATE COURSE
IN
DIABETES AND BASIC METABOLIC PROBLEMS
January 20, 21, 22, 1960 — **Ambassador Hotel**
Los Angeles, California

The Eighth Postgraduate Course in Diabetes and Basic Metabolic Problems will be offered by the American Diabetes Association, January 20, 21, 22, 1960 at the Ambassador Hotel in Los Angeles. The course is accepted for 18 hours credit, Category II, by the American Academy of General Practice. Detailed information is available by writing to American Diabetes Association, Inc., 1 East 45th Street, New York 17, New York.

AMERICAN COLLEGE OF ALLERGISTS
Graduate Instructional Course and Annual Congress
February 28-March 4, 1960 — **The Americana Hotel**
Bal Harbor
Miami Beach, Florida

The American College of Allergists will hold a Graduate Instructional Course and Annual Congress February 28 to March 4, 1960 at the Americana Hotel, Bal Harbor, Miami Beach, Florida. For further information, contact John D. Gillaspie, M.D., Treasurer, 2049 Broadway, Boulder, Colorado.

THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY
March 7-10, 1960 — **Roosevelt Hotel**
New Orleans, Louisiana

The twenty-third annual meeting of The New Orleans Graduate Medical Assembly will be held March 7-10, 1960 in New Orleans. Complete details may be obtained by writing to Mannie D. Paine, Jr., M.D., Secretary, The New Orleans Graduate Medical Assembly, 1430 Tulane Avenue, New Orleans 12, Louisiana.

AMA Names Martin As GP of the Year

ONE OF OKLAHOMA'S most beloved physicians has been selected as the nation's outstanding family physician by the American Medical Association.

Chesley M. Martin, M.D., seventy-year-old Elgin physician, was named the 1959 General Practitioner of the Year by the AMA's House of Delegates during its Thirteenth Clinical Session in Dallas, December 1-4.

The award is given each year to the doctor who exemplifies the collective image of the thousands of family physicians who not only dedicate their lives to their profession, but also exhibit exceptional qualities of community service and good citizenship.

Doctor Martin's accomplishments as a physician and citizen are legendary among the townspeople of Elgin and the surrounding communities as well. Throughout his forty-four years of medical practice, he has unselfishly waged a battle against illness and still found time to serve his profession and his community in countless capacities of leadership.

Chesley Martin's qualities have long been recognized by his many friends and patients. Now, through the efforts of the Comanche-Cotton District Medical Society, physicians and citizens of the United States pay tribute for a job well done.

Gold Medal Presented Before Five Hundred

Doctors Martin and Orr are pictured during the award ceremonies.

Doctor Martin's greatest moment came when Louis M. Orr, M.D., President of the American Medical Association, presented a gold medal and designated him as "America's Family Physician." The event took place before nearly five hundred AMA Delegates and guests at the opening session of medicine's top policy-making body.

On hand to share Doctor Martin's triumph were nineteen proud members of his immediate family. Following a brief visit with well-wishers and a series of press interviews, the surprised physician flew to California for an appearance on the Art Linkletter show.

Shortly after receiving his gold medal, Doctor Martin found time to visit with his wife and a few of their ten grandchildren.

A Big Job in a S

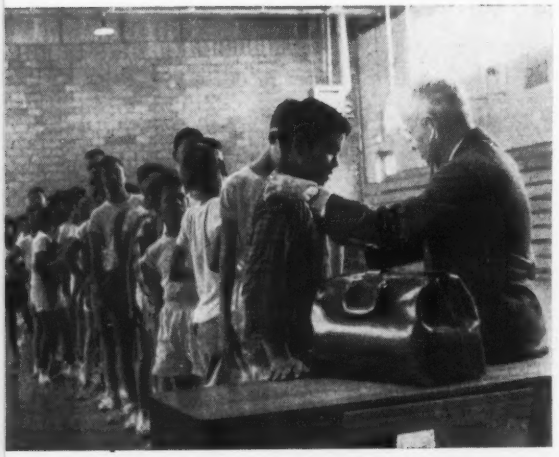
With a work schedule that would try the endurance of a young man, the General Practitioner of the Year has little time for himself. Doctor Martin still spends twelve hours a day, six days a week in the practice of medicine.

The territory he serves is almost boundless, and his able willingness to answer the needs of patients scattered throughout Comanche County has not only endeared him in the hearts of several generations of grateful citizens, but has also resulted in unparalleled respect from his professional colleagues.

A RURAL HOUSE CALL, fifteen miles from Elgin. The physician owns two used automobiles, only to be insured against breakdowns which would immobolize him.

COMANCHE SQUAW, 69 year-old Sally Sovo, receives attention in her home. Doctor Martin cares for twenty-five Indian families who live in the rolling hills around Elgin.

SPRAWLING ELGIN is situated in the ranching country of Southwestern Oklahoma. Thirteen miles north of Lawton, many of its 450 residents hold Civil



PREVENTIVE HEALTH. Thomas Chibitty receives a periodic check up, as do the other five hundred enrollees of the Elgin school system.

OFFICE EXAMINATION is conducted in 31-year-old office on Main Street. The physician also owns and operates the town pharmacy.

in a Small Town

Despite his continuous professional responsibilities, he is the unchallenged "first citizen" of Elgin. The new school buildings, the twelve hundred seat gymnasium, the Future Farmers of America barn and the Methodist Church and parsonage stand as monuments to his accomplishments as a civic leader, architect and builder.

Elgin, Oklahoma found a friend when Doctor Martin came to town in 1916. Its health has been safeguarded, its necessary community facilities have been built and its citizenry has been inspired by this humble, dedicated man!

Service jobs at Fort Sill, while others are ranchers or workers in nearby rock quarries. Large structures in the right foreground are school buildings.

FRIEND TO YOUTH. Doctor Martin is shown examining a prize calf. He helped students get a large barn for FFA work.

MARTIN-DESIGNED Methodist Church is Elgin's most beautiful building. He is shown here listening as Mrs. Harry Leonard practices on the organ he donated.

STANDING IN VAST GYMNASIUM, Martin surverys his work. He designed and supervised the construction of the gym in 1949, at the modest cost of $40,000. It seats twelve hundred.

EDUCATIONAL LEADER. Elgin's physician-School Board Chairman is shown discussing problems with Superintendent Arthur Farrar, Ph.D.

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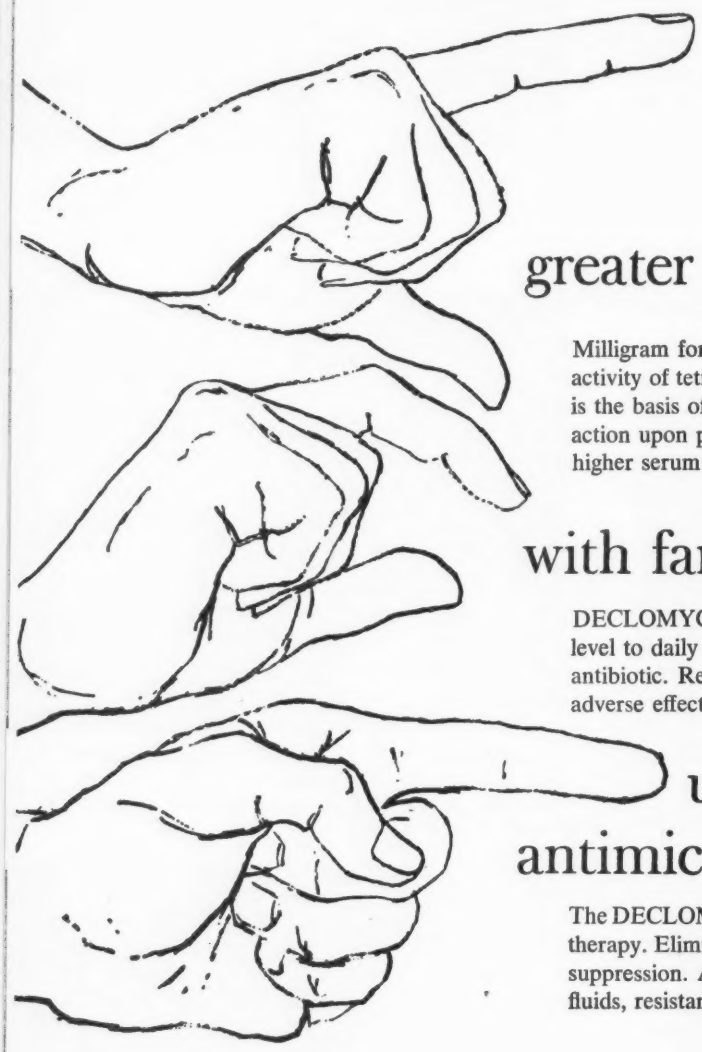
a masterpiece of



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with far less antibiotic intake

DECLOMYCIN demonstrates the highest ratio of prolonged activity level to daily milligram intake of any known broad-spectrum antibiotic. Reduction of antibiotic intake reduces likelihood of adverse effect on intestinal mucosa or interaction with contents.

unrelenting peak antimicrobial attack

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*Hirsch, H. A., and Finland, M. *New England J. Med.* 260:10 (May 28) 19

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COUNCIL LIKES BLUE SHIELD PLAN FOR SENIOR CITIZENS

Culminating nearly a year of investigation and development of an insurance or prepayment plan for the "senior citizen," OSMA's Council, in a special session on Sunday, November 22, approved a Blue Shield "indemnity" proposal, referring it to the House of Delegates for final ratification.

Committee action in this regard has been continuous since early 1959, but the "service plan" approach had thus far prevented Council or Delegates' approval.

Flash Back

Following a request by the American Medical Association (December 1958) that " . . . the AMA, the constituent and component medical societies, as well as physicians everywhere, expedite the development of an effective voluntary health insurance or prepayment program for the group over sixty-five with modest resources or low family income," the Council and the House of Delegates, in April of this year, heard a report from OSMA Committee on Health Care of the Aged which called for the development of such a plan. A more specific request from a joint committee, representing the Blue Shield Laison Committee and the Insurance and Retirement Sub-Committee of the Health Care of the Aged Committee, was also presented and approved. Consequently, Alfred T. Baker, M.D., newly-installed President of OSMA, set the joint group in action to develop a detailed program for the over-sixty-five group.

In a called meeting of the House of Delegates on August 2, the policy-making body disapproved a "service plan" proposal outlined by the joint committee. Principle opposition to the measure came from those who questioned the wisdom of self-committment to a below-par fee schedule. It was the majority feeling that such a concession, even on behalf of the elderly, would inadvertently lead the public to believe that the reduced fees would be an acceptable rate of pay for other special interest groups.

The committee was instructed to continue it study along indemnity lines and to devote its effort to the development of a program for physician services, without assuming responsibility for hospital coverage.

Doctor Baker added several additional physicians to the committee and requested that the group get underway along the lines requested, as soon as possible. After considering many alternatives, the committee requested that the Blue Shield Board simply increase the age limits on the Blue Shield 200 Contract, with corresponding reductions in benefits to permit a realistically priced plan.

Council's Latest Action

The Council's action at the November meeting was in response to the Blue Cross-Blue Shield Boards' reply to the committee request, which was submitted to the Council in the form of the following resolution:

"That the Blue Cross Board of Trustees approve the offering of the regular Blue Cross contract to people sixty (60) years of age and over, except that the number of days' care be limited to thirty (30).

That the Blue Shield Board of Trustees approve the offering of the Blue Shield standard contract ($200 fee schedule) to people sixty (60) years of age and over, except that in-hospital medical care be limited to eighteen (18) days, beginning with the fourth (4th) day.

Further, that these contracts be offered in each county at the next regular non-group enrollment in that county; that the rate for these contracts shall be the same as that charged the regular non-group Blue Cross-Blue Shield members in that county.

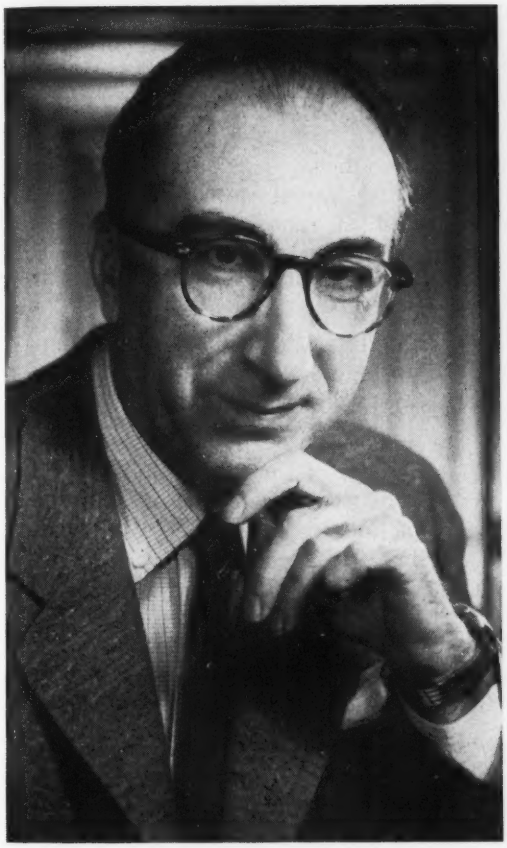
Further, that the experience of holders of these contracts shall be allocated to the rest of the contract holders in that county, both group and non-group, on the basis of contracts in force."

Other Actions

In addition to the Blue Shield proposal, the Council:

(Continued on Page 823)

DeBakey to Appear on Annual Meeting Panel



Michael Ellis DeBakey, M.D., Professor of Surgery and Chairman of the Department of Surgery, Baylor University College of Medicine, Houston, Texas, will appear as a panelist for one of the outstanding features of the scientific program for the OSMA Annual Meeting.

An outstanding man in his field, Doctor DeBakey has won many awards including the Legion of Merit, U.S. Army in 1945, the Rudolph Matas Award in Vascular Surgery in 1954, the AMA Hektoen Gold Medal in 1954, the Modern Medicine Award, in 1949, the International Society of Surgery Distinguished Service Award in 1958, the Roswell Park Medal in 1959 and the AMA Distinguished Service Award in 1959.

Disucssing peripheral cardiovascular diseases with Doctor DeBakey will be Edgar Allen, M.D., Rochester, Minnesota and Robert W. Virtue, M.D., Denver, Colorado. After each speaker discusses the subject, a panel discussion will permit audience participation. Local physicians from related fields will augment the program.

Open Meeting on Public Relations

Following the panel discussion on peripheral cardiovascular diseases which will be held on Tuesday morning, a luncheon meeting will be held in the Hall of Mirrors with Claude Robinson, Ph.D., Chairman of the Board of Opinion Research Corporation, Princeton, New Jersey, as the guest speaker. A national authority on public opinion surveys, Doctor Robinson will present the results of factual research, regarding the image that the medical profession has created in the minds of the public.

Since this subject is one of broader interest than to the physicians themselves, it is anticipated that wives and University of Oklahoma medical students may also be invited to attend.

Six Additional Guest Speakers

Other speakers on the scientific program and their fields of medicine will be: Harold Rosen, M.D., Baltimore, Maryland, psychiatry; Schuyler Kohl, M.D., Brooklyn, New York, obstetrics and gynecology; Vincent Derbes, M.D., New Orleans, Louisiana, dermatology; Ben Felson, M.D., Cincinnati, Ohio, radiology; Harold A. Sofield, M.D., Oak Park, Illinois, orthopedic surgery; and Grayson Carroll, M.D., St. Louis, Missouri, urology.

Specialty Groups to Meet

Another unique feature of the program will be the early afternoon termination of the Monday session, in order to offer related medical societies the opportunity to conduct meetings of their own on Monday afternoon and evening. Several specialty societies plan to use the out-of-state guest speaker of their respective specialty as the principal speaker for a Monday evening banquet.

Oklahoma City's Municipal Auditorium will be the site of OSMA's 54th Annual Meeting on May 2, 3, 4, 1960.

Movie Tags Medical Assistant As "Doctor's PR Ambassador"

The key role played by the physician's medical assistant in creating good public relations is emphasized in a new film now available for showings to medical societies and medical assistants groups.

Entitled "First Contact," the 26-minute dramatic color film shows the mistakes a new office assistant can make unless she is properly trained for her job and points out that medical assistants groups provide opportunities for increasing on-the-job efficiency.

Doctor F. J. L. Blasingame, Executive Vice President of the American Medical Association, who appears in the film, says the medical assistant is a valuable ally in providing better patient care. Says Doctor Blasingame:

"The medical assistant is the doctor's public relations ambassador—often she has first contact with the patient. Her understanding of good doctor-patient relations and her practice of the techniques that create good will are manifold in their value to her physician and the medical profession.

"The professional medical assistant can make it possible for the doctor to concentrate his time on the scientific side of his practice."

In the film Doctor Blasingame comments on the growth of a new national organization, the American Association of Medical Assistants, dedicated to educational improvement and medical profession cooperation. The AMA passed a resolution commending the aims and objectives of the AAMA in December, 1956.

"First Contact" was premiered before 500 members of the American Association of Medical Assistants holding their third national convention in Philadelphia October 16. Prints of the 16 mm film will be available after December 1 to medical societies for showings through AMA's Department of Medical Motion Pictures and Television and to medical assistants groups through the headquarters of the American Association of Medical Assistants in Chicago at 510 North Dearborn Street.

The film, produced for AAMA in cooperation with AMA as a special service of Wyeth Laboratories, also can be obtained by writing directly to the Wyeth Film Library, Box 8299, Philadelphia 1, Pennsylvania.

Medical Sciences Hall of Fame Inducts Twenty-three

Twenty-three persons were inducted into the Oklahoma Medical Sciences Hall of Fame in the second annual installation ceremony sponsored by the Alumni Association of the University of Oklahoma School of Medicine.

Honorees include Mark R. Everett, Ph.D., director and dean of the University of Oklahoma Medical Center, and Louis H. Ritzhaupt, M.D., Guthrie, cited for his service in the state senate.

Others were laymen selected by the physicians for Hall of Fame recognition as having made unique contributions to the advancement of medicine in Oklahoma.

The inductees: Stanley C. Draper, managing director of the Oklahoma City Chamber of Commerce; Nolen Fuqua, Duncan conservation and civic leader; Paul Hoheisel, Oklahoma City, advertising and publicity manager for Oklahoma Gas and Electric Co.; Edith C. Johnson, The Daily Oklahoman columnist; L. D. Lacy, president of Central State Bank of Oklahoma City; Roy Lytle, Oklahoma City attorney;

John E. Mabee, Tulsa oilman and philanthropist; James C. Nance, Purcell veteran lawmaker and newspaper publisher; Howard Neumann, Oklahoma City executive vice-president of the Lowe Runkle Co.; the late W. W. Woodworth, Ardmore business executive, honored posthumously.

Inducted as a group were the 11 members of the Volunteer Women's Service Corps of the Oklahoma Medical Research Foundation, led by Mrs. Erich P. Frank, Oklahoma City. Mrs. Clarence Paine, first chairman and organizer of the corps, came to Oklahoma City from Lansing, Michigan, her present home, for the event.

Ceremonies were during a formal dinner October 17 at the Oklahoma Medical Research Foundation.

February Meeting Scheduled By Oklahoma AAGP

Various fields of medicine and surgery will provide topics for the guest speakers for the 12th Annual Meeting of the Oklahoma Chapter of the American Academy of General Practice to be held in Tulsa, February 15-16, 1960. Site of the two-day meeting will be the Tulsa Hotel.

Seven outstanding guest speakers have been secured for the meeting. They are: Keith Hammond, M.D., Paoli, Indiana; Benjamin Felson, M.D., Cincinnati, Ohio; James Kelley, M.D., Tulsa; Isadore Dyer, M.D., New Orleans, Louisiana; Carle Scuderi, M.D., Chicago, Illinois; A. S. Ridolfo, M.D., Indianapolis, Indiana; and W. D. Snively, Jr., M.D., Evansville, Indiana.

Marshall O. Hart, M.D., Tulsa, is general chairman for the 1960 meeting.

San Antonio Will Host International Medical Assembly

San Antonio will be the site of the 24th Annual Session of the International Medical Assembly of Southwest Texas which will be held January 25-27, 1960 at the Hilton Hotel.

Appearing on the program will be 17 guest lecturers from diversified fields of medicine. They are: Col. Harvey C. Slocum, MC, Washington, D.C., anesthesiology; Louis A. Brunsting, M.D., Rochester, Minnesota, dermatology; Laurance W. Kinsell, M.D., Oakland, California, endocrinology; Bernardo Sepulveda, M.D., Mexico City, gastroenterology; William S. Middleton, M.D., Washington, D. C., internal medicine; Louis G. Welt, M.D., Chapel Hill, North Carolina, internal medicine; Russell N. DeJong, M.D., Ann Arbor, Michigan, neurology; Peter C. Kronfeld, M.D., Chicago, Illinois, ophthalmology; Herbert E. Schmitz, M.D., Chicago, Illinois, obstetrics-gynecology; Leopold G. Koss, M.D., New York, pathology; Alexander S. Nadas, M.D., Baltimore, Maryland, pediatrics; Harold Rosen, M.D., Baltimore, Maryland, psychiatry; John D. Reeves, M.D., Gainsville, Florida, radiology.

Also appearing will be: Wiley F. Barker, M.D., Los Angeles, California, surgery; Wilfred G. Bigelow, M.D., Toronto, Canada, surgery; Robert W. Buxton, Baltimore, Maryland, surgery and John M. Waugh, M.D., Rochester, Minnesota, surgery.

In addition to the scientific program, there will be many social events for the physicians and their wives.

Further information is available by writing to A. O. Severance, M.D., President, 202 West French Place, San Antonio 12, Texas.

Treasury Department Wants Medicare Income Reports

Oklahoma Blue Shield, fiscal administrator of the Dependents' Medical Care Program, recently received information to the effect that the Treasury Department, Internal Revenue Service, has requested that a separate form be filed for each calendar year on physicians to whom Medicare payments in excess of $600 have been made.

The following is quoted from the Revenue Ruling:

"Section 604 (A) of the Internal Revenue Code of 1954 provides, in part, that all persons engaged in a trade or business and making payment in the course of such trade or business to another person, of rent, salaries, wages, premiums, annuities, compensations, remunerations, emoluments, or other fixed or determinable gains, profits, and income (other than certain payments not pertinent here) of $600 or more in any taxable year, . . . shall render a true and accurate return to the Secretary of the Treasury or his delegate, . . . setting forth the amount of such gains, profits, and income, and the name and address of the recipient of such payment."

TB IN OKLAHOMA, 1958
FACT SHEET

Cases on record	*5,848
New cases (1958)	961
Deaths	162
Cases in hospitals	620
Active cases at home	916

*This does not include those in mental hospitals or penal institutions.

Have something to exhibit

If so, you should apply now for space at the 1960 meeting of your association. Scientific Exhibits will again play a major role . . . so why not prepare a graphic presentation of your work and share it with the 700 physicians who will attend the state's largest medical meeting? A research or clinical project may be shown as an individual exhibit, or you may present a single case in the "Klinical Kaleidoscope" section.

Research or clinical project

Perhaps you have done some original research, undertaken some experimental work, or simply drawn some conclusions based upon a series of interesting cases. Then you would be eligible to apply for an exhibit booth of your own in the Scientific Exhibit Section. A subject which has a special visual interest would be particularly suitable . . . and it need not be large—just stimulating!

Bizarre case

All of us have encountered a unique case which is worth talking about. If you have a well-documented one which you would like to share with others, toss your hat in the ring today by completing the application on the opposite page. The "Klinical Kaleidoscope" section will be a varied selection of bizarre cases, each presented with one to five x-rays or illustrations and accompanied by a very brief abstract of the clinical course.

O.S.M.A. ANNUAL MEETING

municipal auditorium · oklahoma city

may 2·3·4, 1960

APPLICATION FOR SCIENTIFIC EXHIBIT SPACE

54th Annual Meeting

OKLAHOMA STATE MEDICAL ASSOCIATION

Municipal Auditorium — Oklahoma City — May 2, 3, 4, 1960

1. **NAME AND ADDRESS OF APPLICANT:**

2. **TITLE OF EXHIBIT:**

3. **INDIVIDUAL BOOTH?** ☐ **"KLINICAL KALEIDOSCOPE" SECTION?** ☐

4. **SPACE REQUIRED:**

   A. **BOOTH: Indicate width in multiples of eight feet** _____

   (All booths will be 6 feet in depth)

   B. **"KLINICAL KALEIDOSCOPE": Indicate square footage:**

   Wall-type display _____ **Table top** _____

5. A. **GENERAL DESCRIPTION:**

   B. **MATERIAL TO BE SHOWN (Subject matter):**

   C. **METHOD OF PRESENTATION (x-ray, photos, graphs, models, etc.):**

6. **PREVIOUS SHOWINGS**

ACT NOW — SPACE IS LIMITED

Applications Must Be Received Prior to March 1, 1960

Oklahoma State Medical Association • Box 9696 • Oklahoma City

BLUE CROSS PERCENTAGE UTILIZATION BY COUNTY

HOSPITAL CARE USED—1958 AVERAGE COMPARED TO AVERAGE FOR FIRST NINE MONTHS OF 1959

County Name	1958	9 mo. 1959	County Name	1958	9 mo. 1959
Adair	122.0%	84.8%	Lincoln	108.5	103.3
Alfalfa	90.1	107.1	Logan	57.1	55.8
Atoka	90.0	98.5	Love	76.4	99.8
Beaver	100.5	86.1	McClain	89.3	96.1
Beckham	101.2	107.5	McCurtain	113.0	119.0
Blaine	115.0	91.7	McIntosh	66.4	93.6
Bryan	70.5	79.5	Major	97.8	100.2
Caddo	97.1	91.5	Marshall	93.8	72.3
Canadian	83.3	79.8	Mayes	92.6	78.8
Carter	92.3	74.6	Murray	69.1	95.2
Cherokee	113.3	88.5	Muskogee	78.4	77.7
Choctaw	106.7	115.2	Noble	98.0	98.9
Cimarron	88.7	131.7	Nowata	120.3	120.1
Cleveland	84.8	79.9	Okfuskee	91.5	71.6
Coal	126.6	104.0	Oklahoma	78.1	70.2
Comanche	89.9	98.5	Okmulgee	95.4	117.5
Cotton	91.6	81.2	Osage	102.3	124.2
Craig	98.4	107.5	Ottawa	101.8	97.8
Creek	109.9	130.9	Pawnee	109.3	109.1
Custer	104.2	80.5	Payne	93.0	87.7
Delaware	100.9	116.6	Pittsburg	103.9	97.2
Dewey	86.7	91.5	Pontotoc	101.0	103.0
Ellis	79.0	85.4	Pottawatomie	84.5	76.8
Garfield	115.0	112.4	Pushmataha	113.0	102.8
Garvin	75.0	84.9	Roger Mills	111.6	135.9
Grady	77.0	75.4	Rogers	96.4	85.9
Grant	106.4	132.2	Seminole	78.5	71.3
Greer	81.5	69.5	Sequoyah	89.7	135.9
Harmon	60.6	64.7	Stephens	97.0	85.9
Harper	90.1	101.0	Texas	118.1	105.7
Haskell	73.3	102.1	Tillman	115.4	108.7
Hughes	106.1	86.5	Tulsa	103.8	101.0
Jackson	82.6	74.7	Wagoner	97.6	76.5
Jefferson	133.5	140.8	Washington	100.5	105.3
Johnston	74.0	68.6	Washita	108.4	68.4
Kay	90.3	89.6	Woods	107.2	99.9
Kingfisher	81.9	67.3	Woodward	91.4	90.8
Kiowa	88.2	90.4			
Latimer	114.8	118.9			
LeFlore	86.9	89.7	Average	94.6%	93.8%

NOTE: Utilization is on a paid basis and does not include reserves, operating expenses, nor any estimated retroactive payments.

Released at the request of the Blue Cross-Blue Shield Liaison Committee for the information and education of OSMA members.

Film on Management of Strokes Issued by Heart Association

A new professsional film designed to acquaint general practitioners and nurses with concepts and techniques basic to the care of stroke patients has been released by the American Heart Association.

Titled "Cerebral Vascular Diseases: The Challenge of Management," the film demonstrates the latest methods of aiding recovery and rehabilitation of stroke patients through use of services and equipment available to all physicians.

A version of this film for lay audiences, titled "Second Chance," has also been produced for the Heart Association and its affiliates. It can be of help to physicians in instructing families of stroke victims on rehabilitation procedures, and as a visual aid in addressing non-professional groups.

Both are 16 mm. black-and-white sound films, produced and directed by George C. Stoney and Associates for the American Heart Association. The films were aided by a grant from E. R. Squibb & Sons, division of Olin Mathieson Chemical Corporation. Prints are available for purchase or loan from the Oklahoma State Heart Association, 825 N.E. 13th Street, Oklahoma City 4, Oklahoma.

Council Likes Blue Shield Plan

(Continued from Page 816)

• Heard comprehensive reports on the Department of Public Welfare medical care program and the Crippled Children's program (now under the direction of the DPW).

• Reaffirmed their appointments to the Crippled Children's Commission Advisory Board.

• Referred a request (Committee on Aging) for the provision of geriatric fellowship funds to a committee for further consideration.

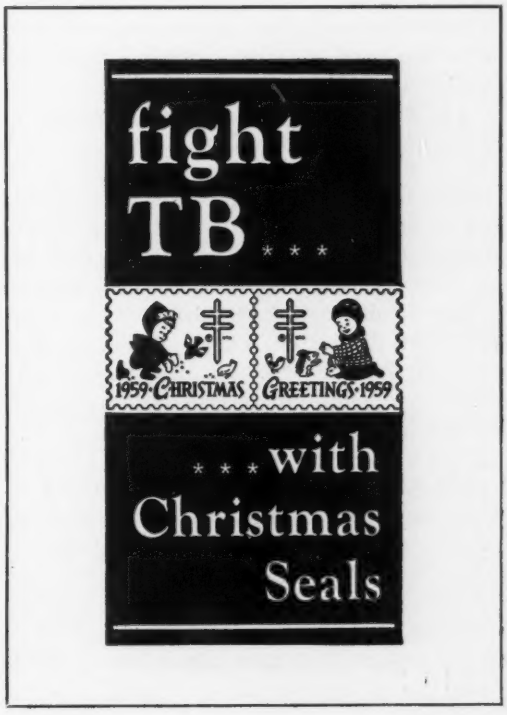
• Approved continued co-sponsorship of a highschool essay contest which is annually promoted by the American Association of Physicians and Surgeons.

State Heart Association Plans February PG Course

Four medical leaders in the field of Cardiac Rehabilitation will be presented for Oklahoma physicians Wednesday, February 10, 1960, under the sponsorship of the Oklahoma State Heart Association's Cardiac in Industry and Agriculture committee.

Scheduled as a post-graduate course at the Oklahoma Medical Center, 800 Northeast 13th Street, the program is designed to bring the latest, most authoritative information on rehabilitation of the cardiac patient.

Out-of-state participants on the program include Frederick J. Kottke, M.D., professor and head of the department of physical medicine and rehabilitation at the University of Minnesota, Minneapolis; Amasa B. Ford, M.D., department of medicine, Western Reserve University, Cleveland, Ohio; L. E. January, M.D., department of internal medicine, University of Iowa, Iowa City; and Carlton D. Chapman, M.D., Professor of Medicine, Southwestern Medical School, Dallas, Texas.



Auxiliary News

Terminal Months

Keen appreciation of terminal months of the year affords us an interesting perspective, as individuals and as Auxiliary members. First of all, we can extend our thankfulness to include our privilege to meet as wives of physicians, women with mutual interests. In the spirit of giving, we can include our greatest potential: individual responsibility in giving of our time and talent.

Further, in respect to the coming of the new year, we can evaluate the effectiveness of our own Auxiliary program in each county, strengthening any weakness which might be apparent thus far in the calendar year.

Time and Talent

As we approach the beginning of a new decade, Auxiliary members are increasingly aware that this period will be crucial—can be critical. In addition to the time and talent needed, our Auxiliary needs continually increased interest and concern. Where there is group interest and concern, necessary group action can logically follow.

High on our list of projects to forestall and prevent federal control is the American Medical Education Foundation. Each of us can help. In our 85 approved medical schools in this country, student tuition covers only 18.2% of the actual cost of training. Endowment income, grants and bequests from individuals and foundations cover part of the deficit.

A.M.E.F. was established in 1951. The AMA pays for all expenses of this worthy project, so that no monies are withheld for operating expenses of the fund. Every dollar contributed goes directly to the medical school of your choice, or to the general fund, if you prefer. Five past presidents of AMA form the governing board of A.M.E.F.

Mrs. Karl F. Ritter, National Chairman, has announced that our contribution for 1958-59 was $140,500, which includes donations from Auxiliaries and from individual members. This year the goal of $175,000 has been set. Mrs. Ritter explains: "If each member accepted the responsibility of providing $5 for A.M.E.F. . . . we would have more than twice the goal we have set for ourselves. Is this asking too much—$5 worth of interest and support over a year's time?"

Mrs. Pat Fite, Sr., State A.M.E.F. Chairman, lists the following as types of gifts that can be made to the fund:

1. Put A.M.E.F. in your Auxiliary budget, if you have not already done so.
2. Use the fund as a means of expressing personal thanks to a doctor.
3. Honor a graduate from a Medical School.
4. Express appreciation to some one who has paid his (or her) own expenses to come to address your meetings.
5. Use as a "thank you" to a hostess.
6. Contribute to A.M.E.F. as a memorial tribute to deceased friends.

Contribution cards are available from your local A.M.E.F. Chairman; or, if you are a member-at-large or your Auxiliary does not have an A.M.E.F. Chairman, send your check with the name and address of the person honored and the specified medical school to:

Mrs. Pat Fite, Sr.
1417 Emporia Street
Muskogee, Oklahoma

What better way to celebrate Thanksgiving and Christmas—and to start the New Year? Send a gift, either memorial or donation (tax deductible) to A.M.E.F. to express our thankfulness and to help further medical freedom!

25 YEARS AGO

Articles published in *The Journal* of the Oklahoma State Medical Association, December, 1934.

Ano-Rectal Fistulae

Raymond L. Murdock, M.D., Oklahoma City

Practically the treatment of fistula in ano is operation. It may be either incision (fistulotomy) or excision (fistulectomy) with or without a variable amount of closure by suturing. The distal part of the tract may be excised and a silk ligature threaded through the remainder encircling the involved part of the sphincter, this latter to be incised at a later time when the distal part has filled in and the granulation will prevent undue retraction of sphincter ends when they are severed. Fistulectomy under antiseptic precautions of a superficial distal tract can be followed by suture of that part of the wound. On the other hand a lateral fistulous tract often goes very deep being the remains of the abscess in the ischio-rectal or supra-levator spaces. This has to be kept open and will heal if the other tract from the ano-rectal junction is laid open and the reinfection thus stopped. Deep incision into the anus can be made in the midline posteriorly. Judgement must be exercised as to the amount of sphincter that can be cut at the other parts of the circumference at a single operation. Other precedures to prevent undue retraction of the cut ends of the sphincter are suturing the ends until granulation tissue sets them in place, and primary suturing of the cut ends to firm tissue underlying them.

I have not had success with the Elting procedure to close the internal opening by mobilization and suture of the bowel wall. However, I will describe a procedure which might be considered akin to it in some respects, which has given me uniformly good results:

Several years ago I began saving the continuity of considerable sphincter muscle at the site of fistulectomy. Previously I had usually followed the practice in rectal surgery of cutting through everything lying between the fistula and the lumen of the bowel. By using small hemostats and scissors I have been able to make a more minute dissection and excision of the tract without severing the overlying sphincter. A slight amount of freeing of the bowel wall above it is done. It is then possible to place the sphincter upward and stitch the rectal wall downward over it fixing the sphincter against solid tissue in a somewhat higher but nevertheless good functioning position. The final result is a more normal anal contour without the notched defect sometimes marked after the usual operation. Since development of this way of saving sphincter I have been able to operate in a single trip to surgery on two separate fistulae on opposite sides of the anus of the same patient who previously would have had to recover from one, and then return in a month or so for another anaesthetic and operation on the other side.

Discussion

Dr. J. A. Walker, Shawnee

This especial problem is entirely new to me, but some years ago I had a condition similar to this in a boy. I was called down to the south end of the county one night, and I went and found this boy about eleven o'clock at night in his home. The abdomen was distended, and he gave a history that he was out in a pasture and fell into a ditch about ten feet deep, and he got out and came home and finished doing up the chores, and then got sick. His father gave him a couple of C. C. pills and salts, etc., and he promptly threw all that up. They called me and I took Dr. Baxter with me and we landed at the home about eleven o'clock at night, and there was this desperately sick boy. They didn't have any light to speak of, but the doctor happened to have an acetylene light and when we lighted the doctor's light all the flies within three hundred miles came into the house. We didn't have anything to smear on the boy's belly, but we found hanging on the wall about an ounce of tincture of iodine and we smeared that on the boy's abdomen and anesthetized him after telling the father there was nothing to do except operate, and we didn't want to do that. We laid him up on the kitchen table and went ahead. Dr. Baxter said: "Here is our kink," and over the right side there was an enlargement and completely obstructed bowel. We picked it up, and there was the intestine perfectly occluded, about twelve or fifteen inches above the ileoceal valve. We got that open and there was a rock about as big as a golf ball, or maybe a little larger. About eight inches above this there was another obstruction, and we slipped that down to the same hole and dropped it out. The boy got well and seems to be well yet.

Dr. Kuhn: He must have absorbed those rocks when he fell in the ditch.

The 1959 Christmas Seal Campaign to fight tuberculosis opened November 16, and continues through December.

Progress in the fight against tuberculosis, since the first Christmas Seal Campaign, has meant a saving of more than 8½ million lives.

PHYSICIAN PLACEMENT

Dermatology

Thomas E. Slimp, M.D., 323 West Livingston Place, Metairie, Louisiana, age 42, married, Syracuse Medical School, 1950, board certified January 1, 1960. No military obligations, available January 1, 1960.

Gordon Harold Ekblad, M.D., Field Medical Service School, Camp Pendleton, California, 53, married, University of Minnesota, 1930, available January 1960.

General Practice

Donold D. Arthurs, 3240 N.W. 26th St., Oklahoma City, Oklahoma, age 27, married, Oklahoma University School of Medicine, 1958, no military obligations, available immediately.

Johnny Bill Delashaw, U.S.P.H.S. Hospital, Wyman Park Drive and 31st Street, Baltimore 11, Maryland, age 27, married, University of Texas Medical Branch, 1959, available July 1960.

D. H. Bessesen, M.D., Grand Canyon Hospital, Grand Canyon, Arizona, age 61, married, University of Minnesota, interested University Health Service, available October 1, 1959.

Sherman Allen Hope, M.D., 1105 Yale Ave., Panama City, Florida, PO 3-6656, age 27, married, University of Oklahoma School of Medicine, 1957, available July 1960.

Paul J. Vega, M.D., 6070 Orleans Ave., New Orleans, Louisiana, age 26, married, Louisiana State University, 1956, no military obligations, available now.

Thomas S. Whitecloud, M.D., 858 Market Street, Pascagoula, Mississippi, age 45, married, Tulane, 1943, veteran, has interest in teaching, availability date depends on situation.

Internal Medicine

Richard I. Hochman, M.D., 37, Badger Road, Annapolis, Maryland, 31, married, New York University, 1952, available October 1, 1960.

Albert D. Roberts, Jr., M.D., 4333 Colgate, Dallas, Texas, 29, married, Southwestern Medical School, Veteran, available July 1959.

Van B. Saye, Jr., M.D., 1230 Brockenbraugh Court, Metairie, Louisiana, age 28, married, Medical College of Georgia, 1954, Air Force Reserve, available July 1, 1960.

Locum Tenens

Arnold Giesbrecht, M.D., Hallock, Minnesota, age 34, married, graduate Winnipeg, Manitoba, 1957, prefers suburban or locality close to cities.

James D. Green, M.D., second year resident in Internal Medicine at St. John's Tulsa, desires one year of Internal Medicine practice before taking third year of training. Prefers clinic in city of 50,000 or greater population.

Russell M. Preston, M.D., 476 San Juan, Oak Harbor, Washington, desires six month's work in General Practice between time of separation from military service to beginning of residency training, January-July, 1960.

Obstetrics and Gynecology

Lynn W. Abshere, M.D., 708 N.E. 14th, Oklahoma City, age 31, married, University of Oklahoma School of Medicine, 1956, American College of OB-GYN, no military obligations, available July 1, 1960.

Gerald R. Keilson, M.D., Medical Arts Building, Dallas, Texas, age 31, married, University of Texas, 1953, board qualified, veteran, available in July 1960.

Milton Gardner Mutch, Jr., M.D., 423 Mills Avenue, Fargo, North Dakota, age 31, married, University of Minnesota, 1955, board eligible upon completion of fellowship, veteran, available July 1, 1959.

David H. Holmes, M.D., 1017 Grovena Drive, St. Louis, Missouri, 37, married, Washington University, St. Louis, Veteran, available July 1, 1960.

Nejdat Mulla, M.D., 1110 Belmont Avenue, Youngstown, Ohio, age 36, married, University of Geneva, Switzerland, 1952, American College of OB-GYN, not eligible military service, available July 1, 1960.

Harold G. Ray, M.D., 212 Darien Place, Box 503, Balboa Heights, Canal Zone, age 28, married, University Arkansas School of Medicine, 1955, now in active reserve, available July 1, 1960.

Alvon C. Winegar, M.D., 1940 Michigan, Benton Harbor, Michigan, age 41, married, Wayne University, 1942, no military obligation, available early 1960.

Pediatrics

H. M. McClintock, M.D., 5712 St. John St., Kansas City, Missouri, 29, married, Baylor University, 1955, veteran, available December 1960.

Surgery

(Name on Request) 32 years old, married, Tulane, 1952, veteran, board eligible.

Johnson P. Graves, M.D., Box 142, Pulaski Heights Station, Little Rock, Arkansas, age 36, married, Arkansas School of Medicine, 1950, board eligible, no military obligations, available Fall 1959.

Olin O. Williams, Jr., M.D., 3768 Alma Drive, Memphis, Tennessee, 29, married, University of Tennessee, veteran, available January 1962.

Urology

Charles Lee Reynolds, Jr., M.D., 5012 Rockport Drive, Dallas, Texas, age 32, Southwestern Medical College, Dallas, 1949, board eligible, married, no military obligations, available July 15, 1960.

Alex Grossman, M.D., V.A. Center, Temple, Texas, 42, married, University of Texas, 1951, board certified, veteran, available now.

Herbert Sohn, M.D., 2010 Cliffview Road, Cleveland. Ohio, 32, married, Chicago Medical School, 1955, veteran, available July 1960.

MISCELLANEOUS ADVERTISEMENTS

17 BED hospital, clinic and equipment for sale. Prefer to sell all to one buyer in attractive package offer, but will consider other offers. Equipment includes X-ray, fluoroscope, Medco Sonalator, BMR, diathermy, etc. In settling estate of Charles W. Ohl, M.D., facilities and equipment will be sold at considerably less than original cost, and time payments may be arranged. Contact Walt Allen, Co-Executor, 116 North 4th Street, Chickasha, Oklahoma.

WANTED: General Practitioner—Five man established group. Prefer locum tenens for one year. Will meet competitive salary requirements for qualified man. Contact H. B. Landholt, Business Manager, Physicians & Surgeons Clinic, Box 711, Holdenville, Oklahoma.

FOR SALE: $40,000 per year General Practice. 14 room completely equipped clinic. County seat town of 4,000 with new 40 bed hospital. $5,000 will handle. Write Key J, The Journal, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

PHYSICIANS WANTED: Qualified Physicians needed to work with psychiatric patients in 2,400 bed hospital near Tulsa; salary range $9,180 to $12,120; if Board Certified $11,700 to $15,540. Citizenship required. Vacationland for fishing, boating, other outdoor sports. Write for other particulars, Eastern State Hospital, Vinita, Oklahoma.

OFFICE SPACE for rent, modern building, 528 N.W. 12th, Oklahoma City. Complete maintenance, air conditioning, free parking space for patients. CE 6-1631.

WANTED: Internist to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

WANTED: Surgeon to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

Internist, Board eligible, 38, family, experienced clinical research, teaching; chief interests cardiovascular, arthritic, geriatric diseases. Prefer Midwest or Southwest individual, small group near medical school. Licensed in Oklahoma. References, curriculum vitae available. Write Key B, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

EXCELLENT OPPORTUNITY in Dallas, Texas for pediatrician to practice in building with five other specialists, including one established pediatrician. This is a group of specialists all having their own individual practice. Contact Key C, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

Complimentary space available to members for three consecutive months. For non-members, the rate is $5.00 each month for each inch of copy or fraction thereof.

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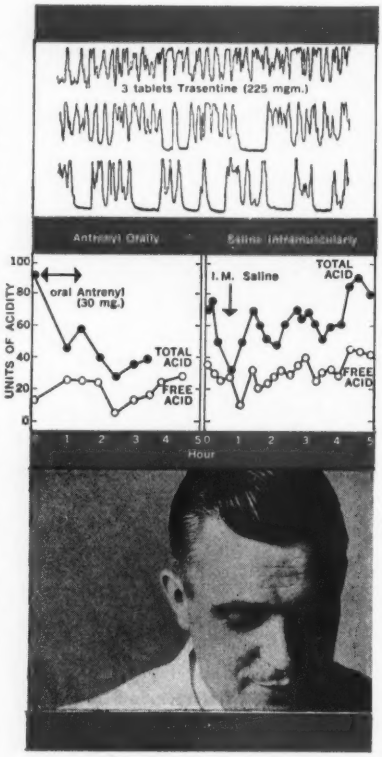

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2. Mattman, P. E., and Strutner, L.: Am. J. Digest. Dis. *20*:126 (May) 1953.

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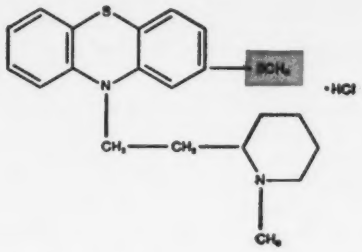

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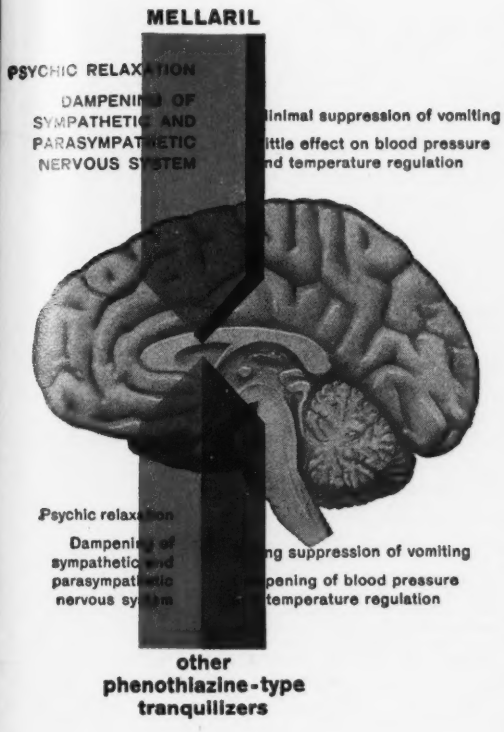

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*Ostfeld, A. M.: Scientific Exhibit, American Academy of General Practice, San Francisco, April 6-9, 1959

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It is generally agreed that it is ideal to withhold antibiotic and chemotherapeutic drugs until after sensitivity tests show which antibacterial agent will be most effective. But very often, in actual practice, the physician knows that delay in starting antibacterial treatment may be detrimental to the welfare of his patient. He must then select the therapy to meet the most serious and immediate threats to the patient.

Why Combination Therapy?

Certain infections do not respond as well to a single agent as to a combination. *Hemophilus influenzae* infections, which are frequent in children, are a particularly serious threat to infants and children up to about 3 or 4 years of age since they have not yet built up any appreciable immunity. Serious complications such as influenzal pneumonia, empyema, or meningitis may develop, especially in this age group. In fact, except for those periods when meningococcal meningitis is epidemic, *H. influenzae* is the most frequent cause of meningitis.[1] This gram-negative organism is highly susceptible both to the tetracyclines and to the sulfonamides. Even in severe infections, therapeutic failure can be virtually eliminated by giving sulfonamides plus tetracycline.[1] These two agents together constitute the treatment of choice, and give better results than either alone.[2]

Sulfonamides remain the drugs of choice for all meningococcal infections, including meningitis. They readily penetrate the blood-brain barrier and pass into the cerebrospinal fluid in good concentrations.[3] In treating overwhelming meningococcal infections, and complicating infections of the upper respiratory tract caused by other organisms, the addition of tetracycline to sulfas can be valuable.[4]

In recent years the sulfonamides have again been prescribed more and more frequently. In certain serious infections, better results can be obtained with a combination of antibiotic and sulfonamide than with either drug alone (e.g., severe pneumococcal pneumonia or pneumococcal meningitis[5]). Furthermore, mixed infections, to which young children are particularly susceptible, often respond only to combination therapy such as tetracycline with sulfonamides (TETREX c T/S).

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References: 1. Alexander, H. E.: The hemophilus group. In: Dubois, R. J.: Bacterial and Mycotic Infections of Man. Ed. 3, Philadelphia, J. B. Lippincott Co., 1958, p. 470ff. 2. Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics. Ed. 2, New York, The Macmillan Co., 1956, pp. 1322-1323. 3. Beckman, H.: Drugs — Their Nature, Action, and Use. Philadelphia, W. B. Saunders Co., 1958, pp. 527-528. 4. Dingle, J. H.: Meningococcal infections. In: Cecil, R. L., and Loeb, R. F.: A Textbook of Medicine. Ed. 9, Philadelphia, W. B. Saunders Co., 1955, p. 196ff. 5. Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics. Ed. 2, New York, The Macmillan Co., 1956, p. 1308.

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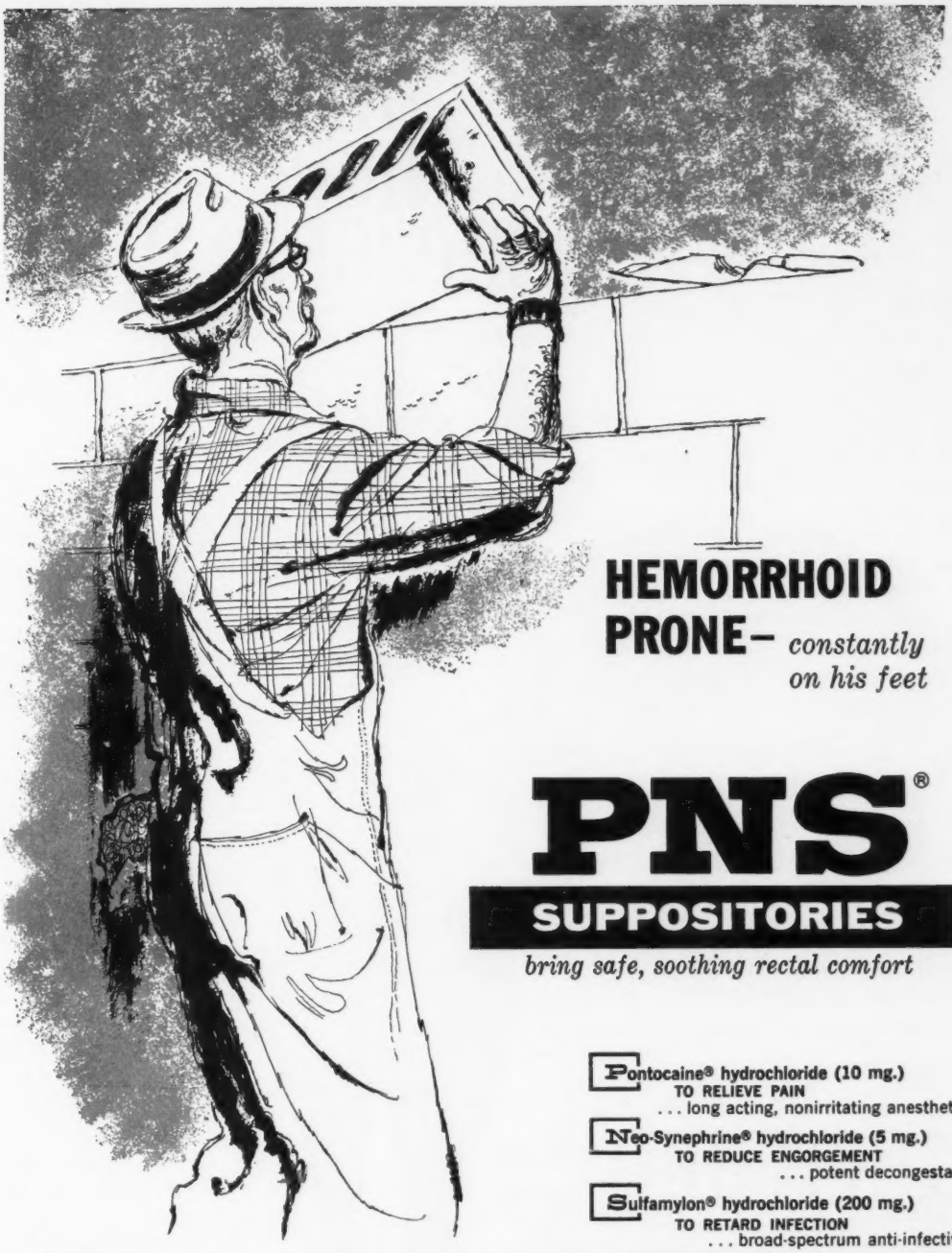

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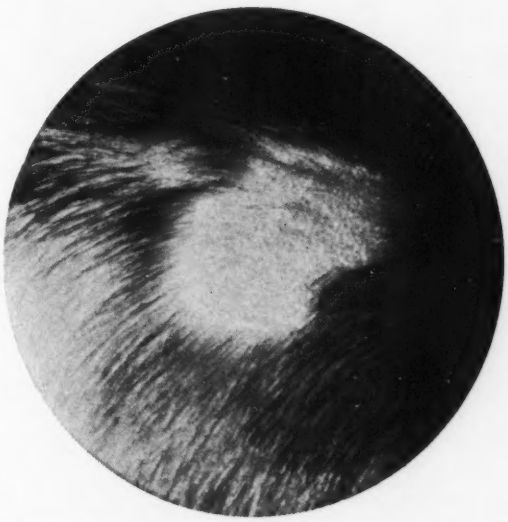

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1. Robinson, H. M., Jr., *et al.*: Griseofulvin, Clinical and Experimental Studies, A.M.A. Arch. Dermat., in press.

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(1) Beck, J. W.; Saavedra, D.; Antell, G. J., & Tejeiro, B.: **Am. J. Trop. Med. 8**:349, 1959.

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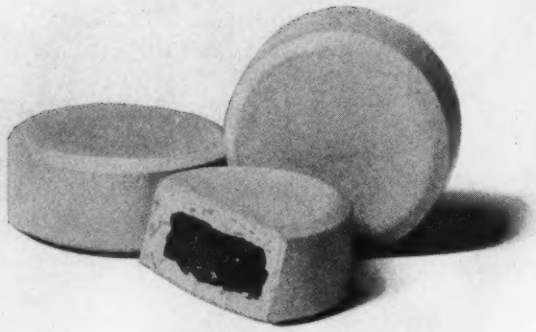

COMPLEMENT FOR KYNEX

Adds fast-acting analgesia of phenylazodiaminopyridine HCl. Relieves burning, urgency and pain-spasm. Eases voiding and retention of infected urine.

... **to unexcelled sulfa control** of KYNEX. Lower dosage of just ½ Gm. daily ... prolonged action without hazard of crystalluria ... reduced toxic potential ... not surpassed by any other sulfa drug, singly or in combination. Dosage: Two tablets q.i.d. first day; one tablet q.i.d. thereafter. Each tablet contains: 125 mg. KYNEX in the shell with 150 mg. phenylazodiaminopyridine HCl in the core.

LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

NEW

DEPINAR

truly repository injectable B_{12} quickly achieves and sustains high B_{12} blood levels for a minimum of 28 days

The Depinar special repository base permits slow absorption from the injection site, thus decreasing the need for frequent administration. Depinar continually bathes the tissues in vitamin B_{12} to provide more effective therapy and make patients feel better longer. A recent clinical report* shows over 98% of Depinar is retained after one week . . . and "Serum level vitamin B_{12} . . . sustained for 28 days or more from the single dose."

Each package of Depinar consists of a multiple dose vial, containing cyanocobalamin zinc tannate (lyophilized) equivalent to 2500 mcg. vitamin B_{12}. The vial of diluent contains 5 cc. Sodium Chloride Solution for Injection. When reconstituted, each ml. of Depinar contains 500 mcg. vitamin B_{12}.

*Thompson, R. E., and Hecht, R. A.: Am. J. Clin. Nutrition 7:311-317 (May-June) 1959.

ARMOUR PHARMACEUTICAL COMPANY • KANKAKEE, ILLINOIS

Armour Means Protection

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ARMOUR

the disease of many masks

Doctor, do you recognize this patient? She complains of flatulence, constipation with alternating periods of diarrhea, and colicky pains in the lower right quadrant. At other times she is troubled by anorexia, lassitude, dull headache, muscle pains and backache. Or she may have only one or two of these symptoms.

In these puzzling cases, serious consideration should be given to intestinal amebiasis—the disease of many masks. Clinicians say it is "one of the most widespread and serious protozoan diseases of man," yet "there is no parasite more often misdiagnosed than is *E. histolytica*." Conservative estimates place the incidence at 10% of the United States population as a whole, and 16% in southern states.

Now Glarubin, a relatively non-toxic amebicide, simplifies the treatment of suspected cases of intestinal amebiasis. Glarubin, a crystalline glycoside from the fruit of *Simarouba glauca*, is a specific amebicidal agent with minimal side effects. It contains no arsenic, bismuth or iodine.

Glarubin is administered orally in tablet form and does not require strict medical supervision or hospitalization. Extensive clinical trials prove it highly effective in intestinal amebiasis, and virtually free of toxicity.

Supplied in bottles of 40 tablets, each tablet containing 50 mg. of glaucarubin. Write for descriptive literature, bibliography, and dosage schedules.

new Glarubin

TABLETS

specific for intestinal amebiasis

THE S. E. MASSENGILL COMPANY

BRISTOL, TENNESSEE

NEW YORK • KANSAS CITY • SAN FRANCISCO

Is one vegetable oil a better cholesterol-depressant than another?

Yes...the one the patient prefers for taste.

No leading vegetable oil can claim superiority over Wesson in its serum cholesterol-depressant effect. As a diet must be eaten to be effective, *the preferred appetite appeal of Wesson* is most important. Through the years, Wesson has been consistently favored over the next selling oil, particularly for flavor (blandness), odor and lightness of color*. Wesson *encourages* the patient to stay on the prescribed diet.

Quality and uniformity you can depend on. Wesson has a poly-unsaturated content better than 50% . Only the lightest cottonseed oils of the highest iodine number are selected for Wesson and no significant variations in standards are permitted in the 22 exacting specifications required before bottling.

Each pint of Wesson contains 437-524 Int. Units of Vitamin E.

Where a poly-unsaturated oil is called for in the diet, Wesson satisfies the most exacting requirements (and the most exacting palates!).

Wesson's Important Ingredients:

Linoleic acid glycerides	50% to 55%
Phytosterol (predominantly beta sitosterol)	0.4% to 0.7%
Total tocopherols	0.09% to 0.12%

Never hydrogenated—completely salt free

*Substantiated by sales leadership for 59 years and reconfirmed by recent tests against the next leading brand with brand identification removed, among a national probability sample.

TRI-AZO-MUL
TRI-AZO-TABS

TRI-AZO-MUL

Each 100 c.c. contains

SULFADIAZINE (microcrystalline)	3.381 Gm.
SULFAMERAZINE (microcrystalline)	3.381 Gm.
SULFAMETHAZINE (Microcrystalline)	3.381 Gm.

In a palatable, stable emulsion pleasantly flavored with True Raspberry Flavor.

Each average teaspoonful (80 min.) represents .5 Gm. (7.7 grs.) of these 3 combined sulfa drugs in suspension.

TRI-AZO-TABS

each tablet contains .5 Gm. (7.7 grs.) of the above three combined sulfa drugs.

Triple Sulfas (Meth-Dia-Mer) Sulfonamides remain unsurpassed among sulfa drugs for Highest potency • Wide Spectrum • Highest blood levels • Safety • Minima side effects.

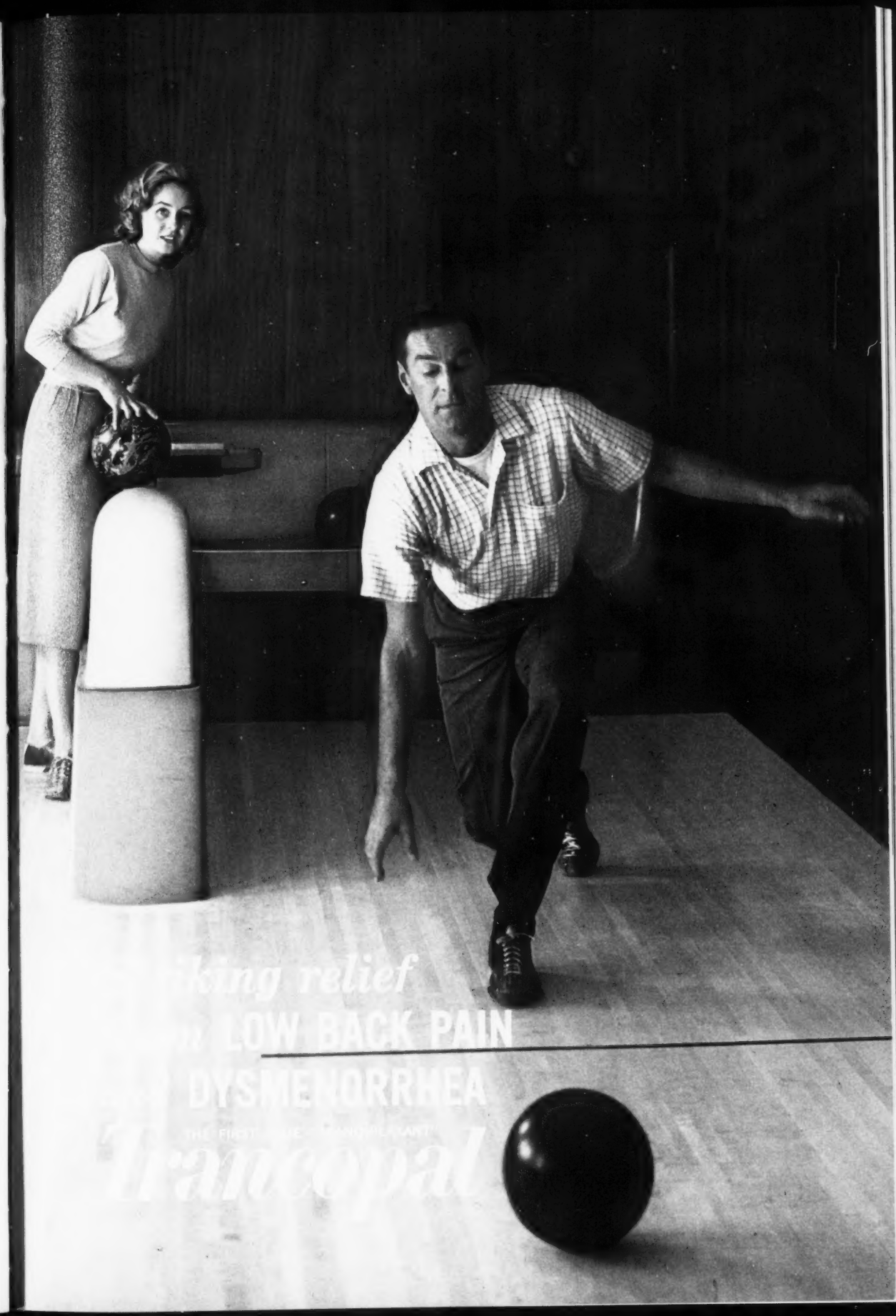
Supplied in pint bottles only.

Tablets in bottles of 100, 500, 1000.

FIRST TEXAS
Chemical Manufacturing Company

1903-1959: Celebrating 56 years of Making FINE PHARMACEUTICALS

1810 N. Lamar **Dallas**


king relief
LOW BACK PAIN
DYSMENORRHEA

Here is what you can expect when you prescribe

for dysmenorrhea

and premenstrual tension

Case Profile*

A 28-year-old married woman, a secretary in a booking agency, complained of severe and consistent pain and cramps in the abdomen during her menstrual periods. Psychologically, she described the first two days as "climbing the walls." Menarche occurred at age 13. She has a regular twenty-eight day menstrual cycle and a four day menstrual period.

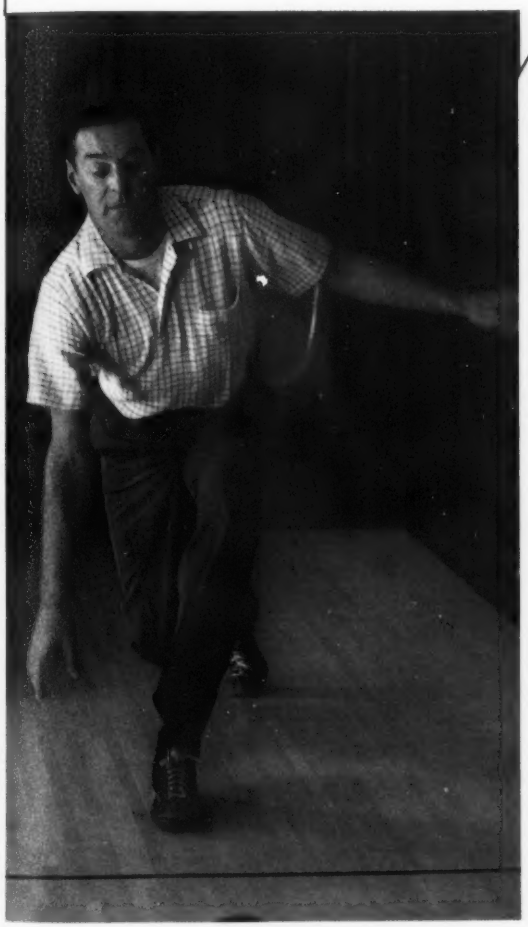
Trancopal was given in a dose of 100 mg. four times a day for the first two days of the four day period. In addition to the relief of the dysmenorrhea she also noticed disappearance of a "bloated feeling" that had previously annoyed her. She has now been treated with Trancopal for one and one-half years with excellent results. Other medication, such as codeine or aspirin with codeine, had relieved the pain, but the patient had had to stay home. Because her father is a physician, many commercial preparations had been tried prior to Trancopal, but no success had been achieved.

Before taking Trancopal this patient missed one day of work every month. For the past year and a half she has not missed a day because of dysmenorrhea.

THE FIRST TRUE "TRANQUILAXANT"

Trancopal®

for low back pain



Case Profile*

A 42-year-old truck driver and mover injured his back while moving a piano. The pain radiated from the sacral region down to the region of the Achilles tendon on the right side. X-rays for ruptured disc revealed nothing pertinent. The day of the injury he was given Trancopal immediately after the physical examination. Although 100 to 200 mg. three times a day were prescribed, the patient on his own responsibility increased the dosage of Trancopal to 400 mg. three times a day. This dosage was continued for three days and then gradually reduced over a ten day period. During this time, the patient continued to drive his truck. The muscle spasm was completely controlled and no apparent side effects were noted.

For the past six months, the patient has continued to take Trancopal 100 to 200 mg. as needed for muscle spasm, particularly during strenuous days.

**Clinical Reports on file at the Department of Medical Research, Winthrop Laboratories.*

Turn page for complete listings of Indications and Dosage.

THE FIRST TRUE "TRANQUILAXANT"

Trancopal

potent MUSCLE RELAXANT

effective TRANQUILIZER

- In musculoskeletal disorders, effective in 91 per cent of patients.[1]
- In anxiety and tension states, effective in 89 per cent of patients.[1]
- Low incidence of side effects (2.3 per cent of patients). Blood pressure, pulse rate, respiration and digestive processes are unaffected by therapeutic dosage. It does not affect the hematopoietic system or liver and kidney function.
- No gastric irritation. Can be taken before meals.
- No clouding of consciousness, no euphoria or depression.

Indications [1-6]

Musculoskeletal:

Low back pain (lumbago, etc.)
Neck pain (torticollis)
Bursitis
Rheumatoid arthritis
Osteoarthritis
Disc syndrome
Fibrositis
Ankle sprain, tennis elbow
Myositis
Postoperative muscle spasm

Psychogenic:

Anxiety and tension states
Dysmenorrhea
Premenstrual tension
Asthma
Angina pectoris
Alcoholism

Now available in two strengths:

Trancopal Caplets®,
100 mg. (peach colored, scored), bottles of 100.

NEW STRENGTH ▶

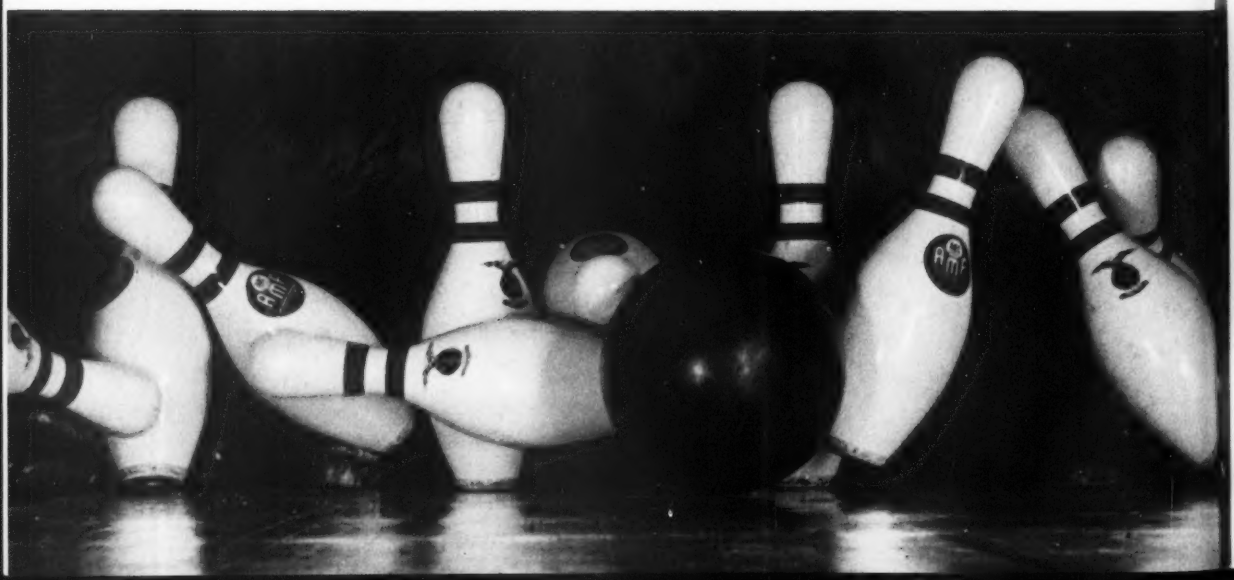
Trancopal Caplets,
200 mg. (green colored, scored), bottles of 100.

Dosage: Adults, 100 or 200 mg. orally three or four times daily. Relief of symptoms occurs in from fifteen to thirty minutes and lasts from four to six hours.

Winthrop LABORATORIES
New York 18, N. Y.

References: 1. Collective Study, Department of Medical Research, Winthrop Laboratories. 2. Lichtman, A. L.: New developments in muscle relaxant therapy, *Kentucky Acad. Gen. Pract. J.* 4:28, Oct., 1958. 3. Lichtman, A. L.: Relief of muscle spasm with a new central muscle relaxant, chlormezanone (Trancopal), Scientific Exhibit, Meeting of the International College of Surgeons, Miami Beach, Fla., Jan. 4-7, 1959. 4. Ganz, S. E.: Clinical evaluation of a new muscle relaxant (chlormethazanone), *J. Indiana M. A.* 52:1134, July, 1959. 5. Mullin, W. G., and Epifano, Leonard: Chlormezanone, a tranquilizing agent with potent skeletal muscle relaxant properties, *Am. Pract. Digest Treat.* 10:1743, Oct., 1959. 6. Shanaphy, J. F.: Chlormezanone (Trancopal) in the treatment of dysmenorrhea; a preliminary report, *Current Therap. Res.* 1:59, Oct., 1959.

Trancopal (brand of chlormezanone) and Caplets, trademarks reg. U.S. Pat. Off. 1408M Printed in U.S.A.



whenever there is inflammation, swelling, pain

VARIDASE®

STREPTOKINASE-STREPTODORNASE LEDERLE

BUCCAL

Tablets

conditions for a fast comeback

as in episiotomy

VARIDASE Buccal provides a simple, natural way to faster, early healing. By activating the fibrinolytic enzymes responsible for normal recovery, VARIDASE shortens the catabolic phase of host response and reverses inflammatory reaction. Edema is reduced.

VARIDASE is not an anti-infective, but by increasing the permeability of the fibrin wall, it eases penetration of natural regenerative factors and fosters healthy tissue growth, making infection less likely.

VARIDASE Buccal Tablets contain: 10,000 Units Streptokinase and 2,500 Units Streptodornase.

Supplied: Boxes of 24 and 100.

Lederle

LEDERLE LABORATORIES,
a Division of American Cyanamid Co.,
Pearl River, New York

For the first time

CONVENIENCE and ECONOMY

for that all-important first dose of broad-spectrum antibiotic therapy

New

TERRAMYCIN®

brand of oxytetracycline

INTRAMUSCULAR SOLUTION

Initiation of therapy in *minutes after diagnosis* with new, ready-to-inject Terramycin Intramuscular Solution provides maximum, sustained absorption of potent broad-spectrum activity.

...and for continued, compatible, coordinated therapy

COSA-TERRAMYCIN®

oxytetracycline with glucosamine

CAPSULES

Continuation with oral Cosa-Terramycin every six hours will provide highly effective antibacterial serum and tissue levels for prompt infection control.

The unsurpassed record of clinical effectiveness and safety established for Terramycin is your guide to successful antibiotic therapy.

Supply:
*Terramycin Intramuscular Solution**
100 mg./2 cc. ampules
250 mg./2 cc. ampules

Cosa-Terramycin Capsules
125 mg. and 250 mg.

Cosa-Terramycin is also available as:
Cosa-Terramycin Oral Suspension — peach flavored, 125 mg./5 cc., 2 oz. bottle
Cosa-Terramycin Pediatric Drops — peach flavored, 5 mg./drop (100 mg./cc.), 10 cc. bottle with plastic calibrated dropper

Complete information on Terramycin Intramuscular Solution and Cosa-Terramycin oral forms is available through your Pfizer Representative or the Medical Department, Pfizer Laboratories.

*Contains 2% Xylocaine® (lidocaine), trademark of Astra Pharmaceutical Products, Inc.

Pfizer

Science for the world's well-being™

PFIZER LABORATORIES, Division, Chas. Pfizer & Co., Inc., Brooklyn 6, N. Y.

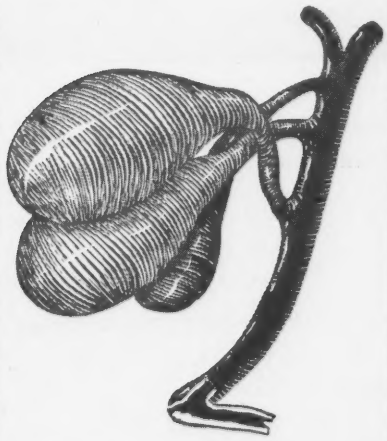
AN AMES CLINIQUICK™

CLINICAL BRIEFS FOR MODERN PRACTICE

HOW PREVALENT ARE MULTIPLE GALLBLADDER ANOMALIES?

One hundred and twenty-two cases of *vesica fellea divisa* (bilobed gallbladder) and *vesica fellea duplex* (double gallbladder with 2 cystic ducts) are reported in the literature. A unique case of *vesica fellea triplex* has recently been described.

Source: Skilboe, B.: Am. J. Clin. Path. *30*:252, 1958.



in medical management and postoperative care of biliary disorders...

"effective" hydrocholeresis...

DECHOLIN®

(dehydrocholic acid, AMES)

"...dehydrocholic acid...does considerably increase the volume output of a bile of relatively high water content and low viscosity. This drug is therefore a good 'flusher,' and is effectively used in treating both the chronic unoperated patient and the patient who has a T-tube drainage of an infected common bile duct."[1]

free-flowing bile
plus reliable spasmolysis

DECHOLIN® WITH BELLADONNA

"...DECHOLIN/Belladonna in a dosage of one tablet t.i.d. for a period of two to three months may prove helpful in relieving postoperative symptoms, aiding the digestion, and facilitating elimination."[2]

(1) Beckman, H.: Drugs: Their Nature, Action and Use, Philadelphia, W. B. Saunders Company, 1958, p. 425.
(2) Biliary Tract Diseases, M. Times *85*:1081, 1957.

AMES COMPANY, INC
Elkhart • Indiana
Toronto • Canada

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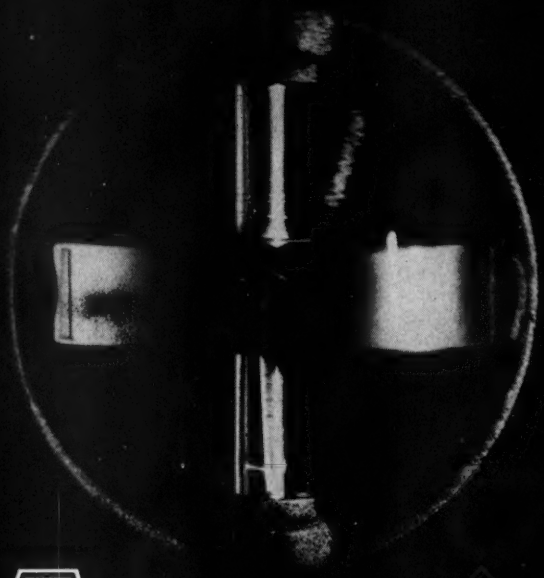
minimal disturbance
of the patient's chemical and psychic balance...



still unsurpassed for total corticosteroid benefits

Arist

Substantiated by published reports of leading clinicians:

- **effective control** of allergic and inflammatory symptoms[1-20]
- **minimal disturbance** of the patient's chemical and psychic balance[1, 4, 5, 8-19]

At anti

-
-
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-
-
-
-

Indication
dermatose
Precautio
served. Do
symptoms.
carried ou
Supplied:
Diacetate

stocort®

Triamcinolone LEDERLE

At anti-inflammatory and antiallergic levels ARISTOCORT means:

- **freedom from salt and water retention**
- **virtual freedom from potassium depletion**
- **negligible calcium depletion**
- **euphoria and depression rare**
- **no voracious appetite—no excessive weight gain**
- **low incidence of peptic ulcer**
- **low incidence of osteoporosis with compression fracture**

Indications: rheumatoid arthritis; arthritis; respiratory allergies; allergic and inflammatory dermatoses; disseminated lupus erythematosus; nephrotic syndrome; lymphomas and leukemias.
Precautions: With ARISTOCORT all traditional precautions to corticosteroid therapy should be observed. Dosage should always be carefully adjusted to the smallest amount which will suppress symptoms. After patients have been on steroids for prolonged periods, discontinuance must be carried out gradually.
Supplied: Scored tablets of 1 mg. (yellow); 2 mg. (pink); 4 mg. (white); 16 mg. (white). Diacetate Parenteral (for intra-articular and intrasynovial injection). Vials of 5 cc. (25 mg./cc.).

References: 1. Feinberg, S.M., Feinberg, A.R., and Fisherman, E.W.: *J.A.M.A.* 167:58 (May 3) 1958. 2. Epstein, J.I. and Sherwood, H.: *Connecticut Med.* 22:822 (Dec.) 1958. 3. Friedlaender, S. and Friedlaender, A.S.: *Antibiotic Med. & Clin. Ther.* 5:315 (May) 1958. 4. Segal, M.S. and Duvenci, J.: *Bull. Tufts North East M. Center* 4:71 (April-June) 1958. 5. Segal, M.S.: Report to the A.M.A. Council on Drugs, *J.A.M.A.* 169:1063 (March 7) 1958. 6. Sherwood, H. and Cooke, R.A.: *J. Allergy* 28:97 (Mar.) 1958. 7. Duke, C.J. and Oviedo, R.: *Antibiotic Med. & Clin. Ther.* 5:710 (Dec.) 1958. 8. McGavack, T.H.: *Clin. Med.* (June) 1958. 9. Freyberg, R.H.; Berntsen, C.A., and Hellman, L.: *Arthritis and Rheumatism* 1:215 (June) 1958. 10. Hartung, E.F.: *J.A.M.A.* 167:973 (June 21) 1958. 11. Hartung, E.F.: *J. Florida Acad. Gen. Pract.* 8:18, 1958. 12. Zuckner, J.; Ramsey, R.H.; Caciolo, C., and Gantner, G.E.: *Ann. Rheum. Dis.* 17:398 (Dec.) 1958. 13. Appel, B.; Tye, M.J., and Leibsohn, E.: *Antibiotic Med. & Clin. Ther.* 5:716 (Dec.) 1958. 14. Kalz, F.: *Canad. M.A.J.* 79:400 (Sept.) 1958. 15. Mullins, J.F., and Wilson, C.J.: *Texas State J. Med.* 54:648 (Sept.) 1958. 16. Shelley, W.B.; Harun, J.S., and Pillsbury, D.M.: *J.A.M.A.* 167:959 (June 21) 1958. 17. DuBois, E.F.: *J.A.M.A.* 167:1590 (July 26) 1958. 18. McGavack, T.H.; Kao, K.T.; Leake, D.A.; Bauer, H.G., and Berger, H.E.: *Am. J. Med. Sc.* 236:720 (Dec.) 1958. 19. Council on Drugs: *J.A.M.A.* 169:257 (Jan. 17) 1959. 20. Rein, C.R.; Fleischmajer, R., and Rosenthal, A.R.: *J.A.M.A.* 165:1821 (Dec. 7) 1957.

LEDERLE LABORATORIES, A Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

in one preparation

the answer to your three most important requirements in a douche

For a dependable and effective means of treating non-specific leukorrhea

For adjunctive therapy in Trichomonas Vaginalis vaginitis and other specific infections

For personal cleanliness and the prevention of irritation and inflammation

TRICHOTINE is the first major douche to contain sodium lauryl sulfate, a detergent of the highest order of efficiency. TRICHOTINE penetrates and dissolves the viscid film covering the vaginal mucosa; gets down in the rugal folds, carrying medication directly to the mucosa and the invading organisms.

TRICHOTINE is a potent bacteri-cide and fungicide, penetrating the walls

of many solution Trichon cans, a potent times ca non-spe

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TRICHOTINE®

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of many micro-organisms. "The douche solution is an effective agent against Trichomonas Vaginalis, Monilia Albicans, anaerobic organisms including a potent strain of streptococci that sometimes cause severe infections, and other non-specific vaginal micro-organisms."[1]

TRICHOTINE actually favors epithelial growth and healing, and the relief it affords from pruritis is quite striking.

For personal cleanliness, especially as a post-coital and post-menstrual douche, TRICHOTINE is designed to meet all the requirements of feminine hygiene. As an effective cleanser for office use, or for treatment, or for routine home douching, TRICHOTINE will prove satisfactory to you and its soothing, refreshing action will be reassuring to your patients. 1. Karnaky, K.J.: Med. Record and Annals, Houston 46:296 (Nov. 1952).

The Fesler Company, Inc., 375 Fairfield Avenue, Stamford, Conn.

TRICHOTINE®

TRICHOTINE®

CHOICE THERAPY FOR THE "OLDER" PATIENT WITH MILD TO MODERATE HYPERTENSION

℞ Veratrite®

More than 13,000,000 prescriptions attest that Veratrite continues to be the antihypertensive of choice for the older hypertensive patient. Veratrite can be prescribed safely and routinely for those who usually cannot tolerate more potent drugs.

Veratrite now contains cryptenamine which acts centrally to produce a gradual fall in blood pressure, yet improves circulation to vital organs, relieves dizziness and headache, and imparts a distinct sense of well-being. Furthermore, Veratrite achieves its effects with unusual safety and without annoying side effects.

Each Veratrite tabule contains: Cryptenamine (tannates), 40 C.S.R.* Units; Sodium nitrite, 1 gr.; Phenobarbital, ¼ gr. Dosage: 1–2 tabules t.i.d., preferably 2 hours after meals.

*Carotid Sinus Reflex

Meisler IRWIN, NEISLER & CO. • DECATUR, ILLINOIS

QUESTIONS ON THE CLINICAL USE OF KANTREX

Q Why should I use KANTREX® Injection* when there are so many other antibiotics available?

A

Because KANTREX Injection is bactericidal to a wide variety of organisms, including many that are highly resistant to the other antibiotics[3,4,10,12,13,17,18,20,21,23,24,25,27,30,33,35,37]

—organisms such as *Staph. aureus, Staph. albus, A. aerogenes, E. coli, H. pertussis, K. pneumoniae, Neisseria* sp., *Shigella, Salmonella* and many strains of *B. proteus.*

Q *But if I use* KANTREX *Injection, won't that help make bacteria resistant to it also?*

Next page, please .....

* Kanamycin sulfate injection (Bristol)

Q *But if I use* KANTREX *Injection, won't that help make bacteria resistant to it also?*

A A very good question, but it is reassuring to note that in almost two years of clinical use of KANTREX for the treatment of infections for which it is recommended, the emergence of KANTREX-resistant bacterial populations has not been a problem.

Q *My impression is that* KANTREX *is just another neomycin. Isn't that so?*

A Indeed not. The only thing KANTREX and neomycin have in common is a similar antimicrobial spectrum. Otherwise, they're very different: they have different chemical structures; the toxicity of KANTREX is "much less than that of neomycin"[14]; and clinically, KANTREX Injection is practical for systemic administration routinely, while neomycin is not.

Q *You mean that* KANTREX *Injection doesn't have the nephrotoxicity of neomycin?*

A Precisely. It's true that when KANTREX Injection is used, urinary casts – even slight albuminuria or microscopic hematuria – may appear, especially in poorly hydrated patients, but this does not reflect any progressive damage to the kidneys. These signs promptly disappear on adequate hydration or termination of therapy.

Q *Then why do you recommend reduced dosage in patients with renal impairment?*

A Because renal impairment causes an excessive accumulation of KANTREX in the blood and tissues, when usual doses are administered. Since KANTREX Injection is excreted entirely by the kidneys, renal impairment leads

to unnecessarily high and prolonged blood levels; and such excessive concentrations increase the risk of ototoxicity.

Q *Is that why we see reports of patients developing hearing loss during* KANTREX *Injection therapy?*

A Yes. A study of the few reported cases in which patients have suffered impaired hearing will show that in every instance they had pre-existing or concurrent renal impairment, yet received usual or excessive doses of KANTREX Injection. Dosage recommendations for KANTREX Injection emphasize that in patients with renal dysfunction, adequate serum levels can be achieved with a fraction of the dose suggested for patients with normal kidney function – with minimal risk of ototoxicity.

Q *Since urinary tract infections are often accompanied by renal impairment, does that mean I shouldn't use* KANTREX *Injection in such conditions?*

A Not at all. With proper precautions, KANTREX Injection is an excellent drug for the treatment of urinary tract infections, especially those due to *Proteus, A. aerogenes* and *E. coli,* even when renal impairment is present.

Q *What are the "proper precautions" in a patient with impaired renal function?*

A The package literature covers them in detail. First, the daily dose should be reduced in such a patient. Then, if he is going to receive KANTREX Injection for 7 days or more, a pre-treatment audiogram should be done, and it should be repeated at appropriate intervals during therapy. If tinnitus or subjective hearing loss develops, or if followup audiograms show significant loss of high frequency response, KANTREX therapy should be discontinued. However, therapy for 7 days or more

is seldom required because the clinical response to KANTREX Injection is so rapid.

Q *Why do you put so much emphasis on* KANTREX'S *"rapid action"? Every antibiotic I've heard about is supposed to be "rapid acting."*

A There is such an abundance of clinical evidence about "rapid acting" that it takes KANTREX Injection out of the "supposed-to" class.[1,2,3,7,8,9,11,15,16,19,21,22,26,29,32,33] Remember, the effectiveness of KANTREX Injection therapy can usually be appraised in 24 to 36 hours. That's definite evidence of rapid action. In fact, one group of investigators reported that "the rapidity with which bacteria are killed by this agent is reflected by the promptness of the clinical response."[29]

Q *Does* KANTREX *Injection cause blood dyscrasias?*

A In extensive clinical and toxicity studies by numerous investigators, as well as almost two years of general use, not a single instance of such toxicity has been reported.

Q *Can I administer* KANTREX *Injection in any other way than by the intramuscular route?*

A Yes. While it's usually given intramuscularly, other routes are practicable: intravenous, intraperitoneal, by aerosol, and as an irrigating solution. Complete instructions are included in the package insert.

Q *So you think I ought to use* KANTREX *Injection as my first choice antibiotic in staph and gram-negative infections?*

A Yes – because all evidence to date indicates that it is bactericidal against a wide range of organisms... rapid acting... does not encourage development of bacterial resistance... is well tolerated in specified dosage... and has not caused any blood dyscrasias.

KANTREX® CAPSULES

for local gastrointestinal therapy... not for systemic infections

Q *Why can't I use* KANTREX *Capsules for systemic medication?*

A Because there is only negligible absorption of KANTREX from the gastrointestinal tract.[3,5,6,8,28,34] Thus, capsules cannot provide effective blood levels.

Q *Then what are* KANTREX *Capsules used for?*

A Preoperative bowel sterilization, and local treatment of intestinal infections due to kanamycin-sensitive organisms.

Q *I've been using neomycin for preoperative bowel sterilization. Why should I switch to* KANTREX *Capsules?*

A Because KANTREX has been rated as "superior to neomycin" for this purpose.[6] It provides rapid and satisfactory control of coliforms, clostridia, staphylococci and streptococci; yeasts do not proliferate; stool concentrations of the drug are exceptionally high; and nausea, vomiting or intestinal irritation have not been observed.[5,6]

Q *What advantages do* KANTREX *Capsules offer me in the treatment of intestinal infections?*

A A high degree of effectiveness against most of the pathogens responsible for such infections: *Salmonella, Shigella, Staph. aureus, E. coli* and *Endamoeba histolytica.* Moreover, their use has been "remarkably free of any side effects."[31]

KANTREX®

INJECTION KANAMYCIN SULFATE INJECTION

INDICATIONS

Infections due to kanamycin-sensitive organisms, particularly staph or "gram-negatives": genito-urinary infections; skin, soft tissue and post-surgical infections; respiratory tract infections; septicemia and bacteremia; osteomyelitis and periostitis.

DOSAGE: INTRAMUSCULAR ROUTE

Recommended daily dose is 15 mg. per kg. of body weight, in 2 to 4 divided doses.

For intramuscular administration, KANTREX Injection should be injected deeply into the upper outer quadrant of the gluteal muscle.

TOXICITY

When the recommended precautions are followed, the incidence of toxic reactions to KANTREX is low. In well hydrated patients under 45 years of age with normal kidney function, receiving a total dose of 20 Gm. or less of KANTREX, the risk of ototoxic reactions is negligible.

In patients with renal disease and impaired renal function, the daily dose of KANTREX should be reduced in proportion to the degree of impairment to avoid accumulation of the drug in serum and tissues, thus minimizing the possibility of ototoxicity. In such patients, if therapy is expected to last 7 days or more, audiograms should be obtained prior to and during treatment. KANTREX therapy should be stopped if tinnitus or subjective hearing loss develops, or if audiograms show significant loss of high frequency response.

OTHER ROUTES OF ADMINISTRATION

KANTREX should be used by intravenous infusion only when the intramuscular route is impracticable. KANTREX can also be employed for intraperitoneal use, aerosol treatment, and as an irrigating solution. See package insert for directions.

PRECAUTIONS

Use of antibiotics may occasionally result in overgrowth of non-sensitive organisms. If superinfection appears during therapy, appropriate measures should be taken.

SUPPLY

Available in rubber-capped vials as a ready-to-use sterile aqueous solution in two concentrations (stable at room temperature indefinitely):

KANTREX Injection, 0.5 Gm. kanamycin (as sulfate) in 2 ml. volume.
KANTREX Injection, 1.0 Gm. kanamycin (as sulfate) in 3 ml. volume.

CAPSULES (for local gastrointestinal therapy; not for systemic medication)

INDICATIONS AND DOSAGE

For preoperative bowel sterilization: 1.0 Gm. (2 capsules) every hour for 4 hours, followed by 1.0 Gm. (2 capsules) every 6 hours for 36 to 72 hours.

For intestinal infections: Adults: 3.0 to 4.0 Gm. (6 to 8 capsules) per day in divided doses for 5 to 7 days. Infants and children: 50 mg. per kg. per day in 4 to 6 divided doses for 5 to 7 days.

PRECAUTION

Preoperative use of KANTREX Capsules is contraindicated in the presence of intestinal obstruction. Although only negligible amounts of KANTREX are absorbed through intact intestinal mucosa, the possibility of increased absorption from ulcerated or denuded areas should be considered.

SUPPLY

KANTREX Capsules, 0.5 Gm. kanamycin (as sulfate), bottles of 20 and 100.

REFERENCES:

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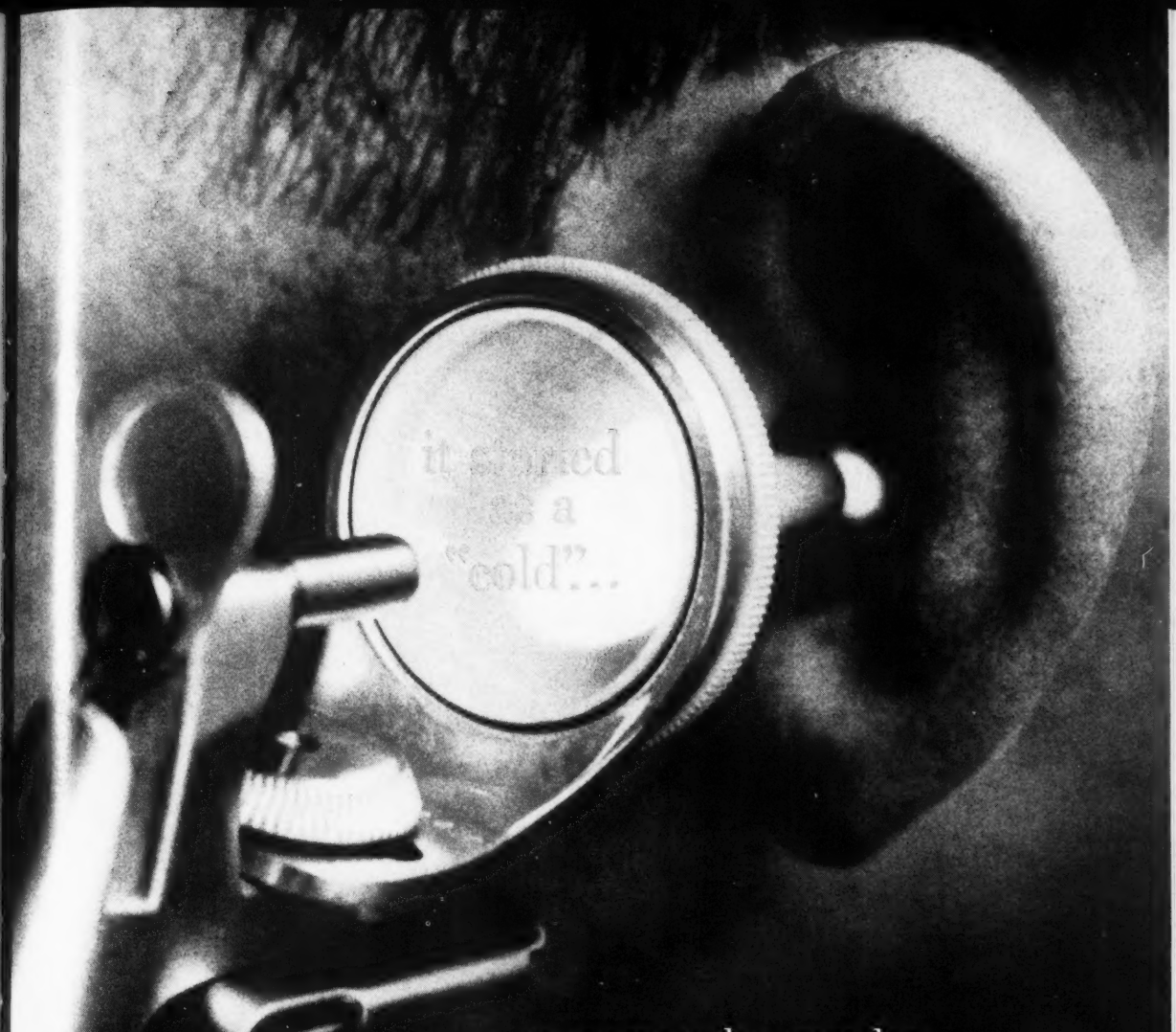


to prevent the sequelae
of u.r.i. ... and relieve the
symptom complex

ACHROCIDIN®

Tetracycline-Antihistamine-Analgesic Compound Lederle

Otitis, tonsillitis, adenitis, sinusitis, bronchitis or pneumonitis develops as a serious bacterial complication in about one in eight cases of acute upper respiratory infection.[1] To protect and relieve the "cold" patient... ACHROCIDIN.

Usual dosage: 2 tablets or teaspoonfuls q.i.d. (equiv. 1 Gm. tetracycline). Each TABLET contains: ACHROMYCIN® Tetracycline (125 mg.); phenacetin (120 mg.); caffeine (30 mg.); salicylamide (150 mg.); chlorothen citrate (25 mg.). Also as SYRUP (lemon-lime flavored); caffeine-free.

1. Based on estimate by Van Volkenburgh, V. A., and Frost, W. H.: Am. J. Hygiene 71:122 (Jan.) 1933

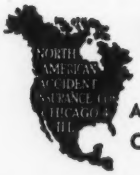
Lederle LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

Season's Greetings

As the underwriters of your

OKLAHOMA STATE MEDICAL ASSOCIATION
DISABILITY INCOME INSURANCE PROGRAM

we wish to take this opportunity to extend warmest Season's Greetings and to wish for you the Merriest of Christmases and the Happiest and Most Prosperous of New Years.



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hydroxyzine pamoate

takes him off the tension treadmill

By restoring tranquility, VISTARIL rapidly helps to relieve functional pain and discomfort in many gastrointestinal disorders. Clinicians find that patients on VISTARIL more willingly accept their condition and adhere better to their regimen.

VISTARIL has an outstanding record of safety and is valuable adjunctive therapy in home or hospital when administered to patients with peptic ulcer, gastroenteritis, esophageal spasm, and nervous dyspepsia.

A Professional Information Booklet is available from the Medical Department on request.

Supply: Capsules—25, 50 and 100 mg.; Parenteral Solution—10 cc. vials and 2 cc. Steraject® Cartridges, each cc. containing 25 mg. hydroxyzine HCl.

Pfizer *Science for the world's well-being™*

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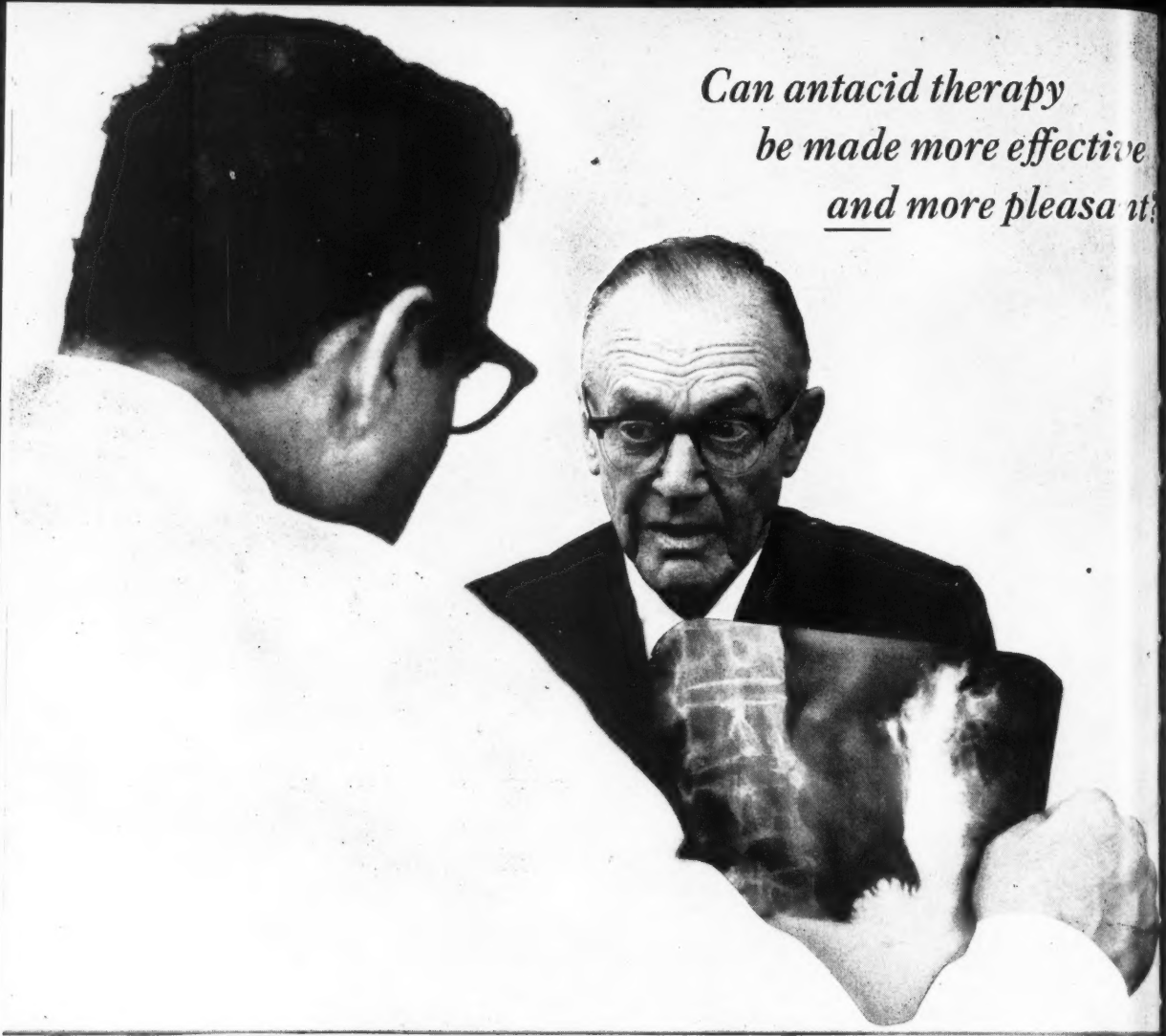

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Creamalin ANTACID TABLETS

Each Creamalin Antacid Tablet contains 320 mg. specially processed, highly reactive, short polymer dried aluminum hydroxide gel, (stabilized with hexitol), with 75 mg. magnesium hydroxide

1. *Neutralizes acid faster (quicker relief)*
2. *Neutralizes more acid (greater relief)*
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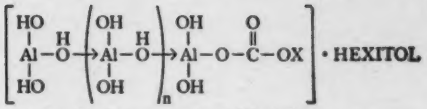
4. *No constipation · No acid rebound*
5. *More pleasant to take*

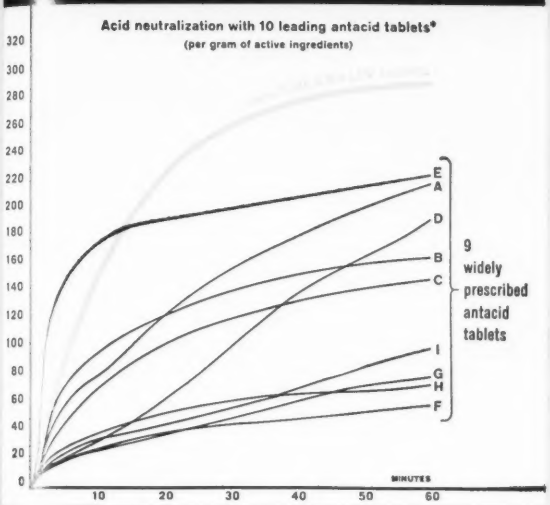
a new high in effectiveness and palatability



n is at least 1 and averages less than 6. X is a cation.

CREAMALIN NEUTRALIZES *MORE* ACID *FASTER*

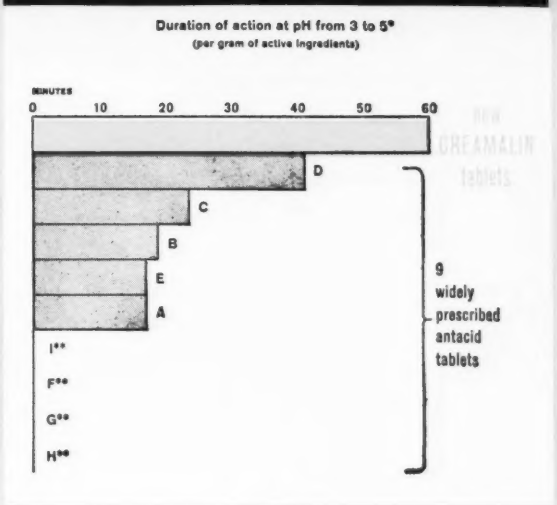
Quicker Relief · Greater Relief



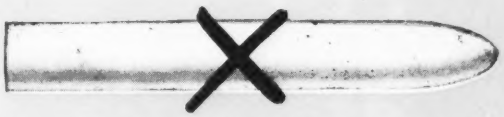
Tablets were powdered and suspended in distilled water in a constant temperature container (37°C) equipped with mechanical stirrer and pH electrodes. Hydrochloric acid was added as needed to maintain pH at 3.5. Volume of acid required was recorded at frequent intervals for one hour.

CREAMALIN NEUTRALIZES *MORE* ACID *LONGER*

More Lasting Relief



*Hinkel, E. T., Jr., Fisher, M. P. and Tainter, M. L.: A new highly reactive aluminum hydroxide complex for gastric hyperacidity. To be published.
**pH stayed below 3.



No chalky taste. New CREAMALIN tablets are not chalky, gritty, rough or dry. They are highly palatable, soft, smooth, easy to chew, mint flavored.

• NO ACID REBOUND • NO CONSTIPATION
• NO SYSTEMIC EFFECT

Adult Dosage: Gastric hyperacidity: 2 to 4 tablets as necessary. Peptic ulcer or gastritis: 2 to 4 tablets every two to four hours. Tablets may be chewed, swallowed with water or milk, or allowed to dissolve in the mouth.

Supplied: Bottles of 50, 100, 200 and 1000.

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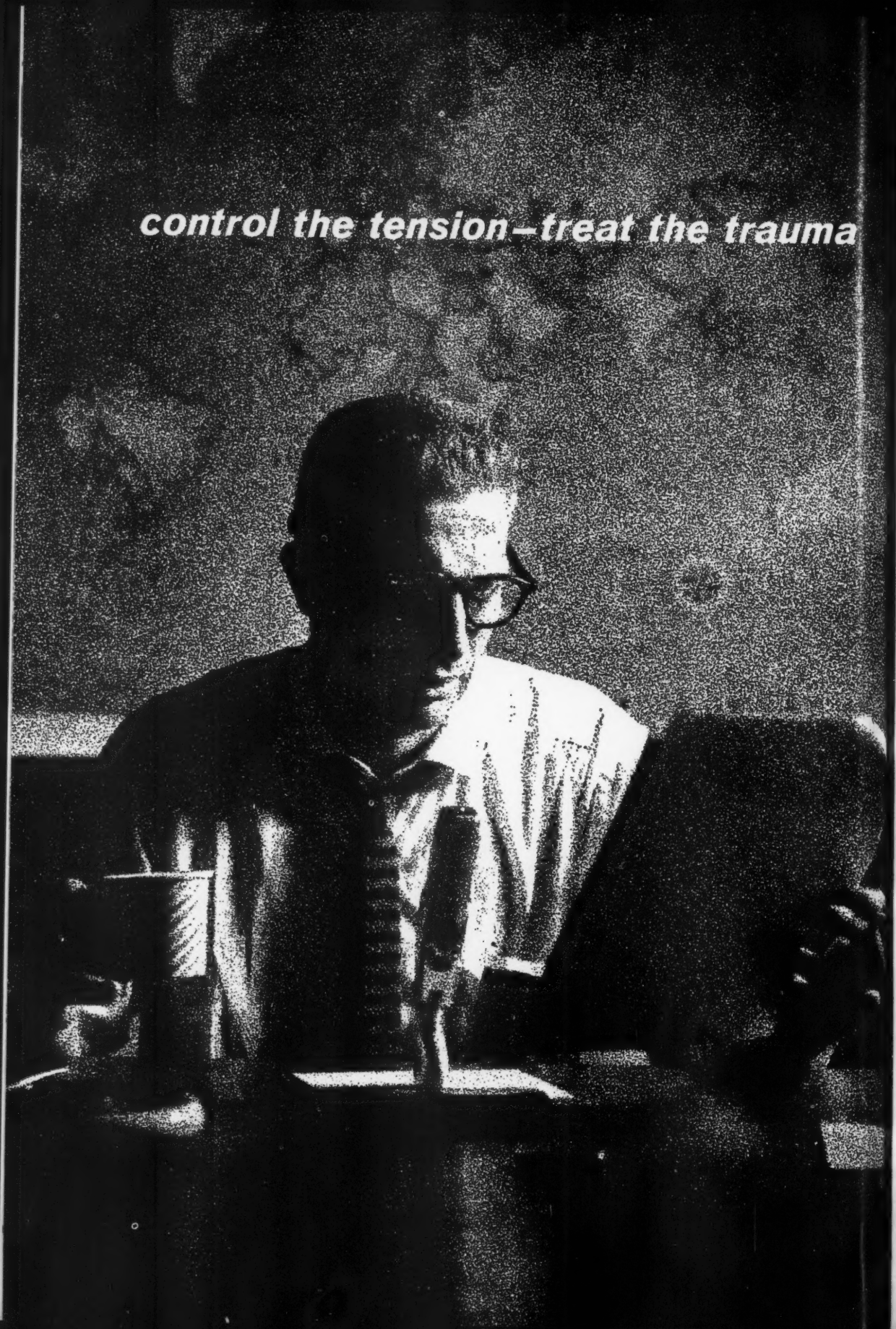
control the tension–treat the trauma

...Pathibamate® 400 200

meprobamate with PATHILON® tridihexethyl chloride Lederle

greater flexibility in the control of tension, hypermotility and excessive secretion in gastrointestinal dysfunctions

PATHIBAMATE combines two highly effective and well-tolerated therapeutic agents:

meprobamate (400 mg. or 200 mg.) widely accepted tranquilizer and . . .

PATHILON (25 mg.)—anticholinergic noted for its peripheral, atropine-like action, with few side effects.

The clinical advantages of PATHIBAMATE have been confirmed by nearly two years' experience in the treatment of duodenal ulcer; gastric ulcer; intestinal colic; spastic and irritable colon; ileitis; esophageal spasm; anxiety neurosis with gastrointestinal symptoms and gastric hypermotility.

Because of individual variation in the intensity of stimuli in gastrointestinal disorders, adequate dosage for optimum control may be expected to vary as well. The dosage strengths of PATHIBAMATE-400 and PATHIBAMATE-200 facilitate individualization of treatment in respect to both the degree of tension and associated G.I. sequelae, as well as the response of different patients to the component drugs.

Supplied: PATHIBAMATE-400—Each tablet (yellow, 1/2-scored) contains meprobamate, 400 mg.; PATHILON tridihexethyl chloride, 25 mg.
PATHIBAMATE-200—Each tablet (yellow, coated) contains meprobamate, 200 mg.; PATHILON tridihexethyl chloride, 25 mg.

Administration and Dosage: PATHIBAMATE-400—1 tablet three times a day at mealtime and 2 tablets at bedtime.
PATHIBAMATE-200—1 or 2 tablets three times a day at mealtime and 2 tablets at bedtime.
Adjust to patient response.

Contraindications: glaucoma; pyloric obstruction, and obstruction of the urinary bladder neck.

Lederle

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NIAMID*

brightens life for the aged

NIAMID gives the depressed elderly person a new sense of well-being. The family will notice a sunnier outlook, an alert interest in group activities, a renewed awareness of personal appearance, and a return of appetite. Your patient will be more cooperative and less demanding.

You can expect to see the same excellent response to NIAMID in a wide variety of depressive syndromes—acute or chronic, mild or severe, whether associated with long-standing or incurable illness, or masquerading as organic disease.

NIAMID side effects are infrequent and mild, and often lessened or eliminated by a reduction in dosage. NIAMID has not been reported to cause jaundice, and significant hypotensive effects have rarely been noted.

DOSAGE: Start with 75 mg. daily in single or divided doses, and adjust according to patient response. NIAMID acts slowly, without rapid jarring of physical or mental processes. Some patients respond to NIAMID within a few days, but for full therapeutic benefit, most require at least two weeks. NIAMID is available as 25 mg. (pink) and 100 mg. (orange) scored tablets.

Already clinically proved in several thousand patients—

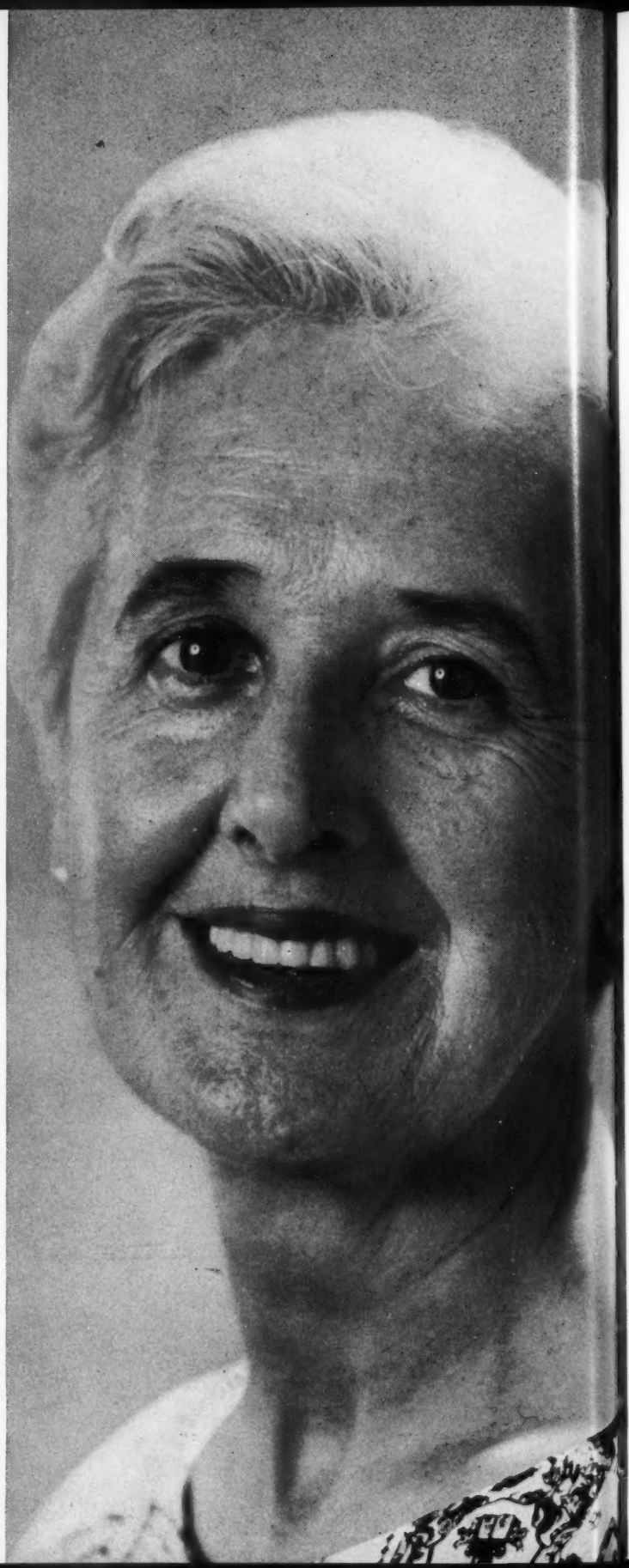
Complete references and a Professional Information Booklet giving detailed information on NIAMID *are available on request from the Medical Department, Pfizer Laboratories, Division, Chas. Pfizer & Co., Inc., Brooklyn 6, N. Y.*

NIAMID

the mood brightener in geriatrics

*Trademark for nialamide

Pfizer *Science for the world's well-being™*



for the tense and nervous patient

relief comes fast and comfortably

–does not produce autonomic side reactions
–does not impair mental efficiency, motor control, or normal behavior.

Usual Dosage: One or two 400 mg. tablets t.i.d.

Supplied: 400 mg. scored tablets, 200 mg. sugar-coated tablets or as MEPROTABS*—400 mg. *unmarked,* coated tablets.

Miltown®

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NOW

. . . a new way to relieve pain and stiffness in muscles and joints

indicated in:

MUSCLE STIFFNESS

LUMBOSACRAL STRAIN

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WHIPLASH INJURY

BURSITIS

SPRAINS

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FIBROSITIS

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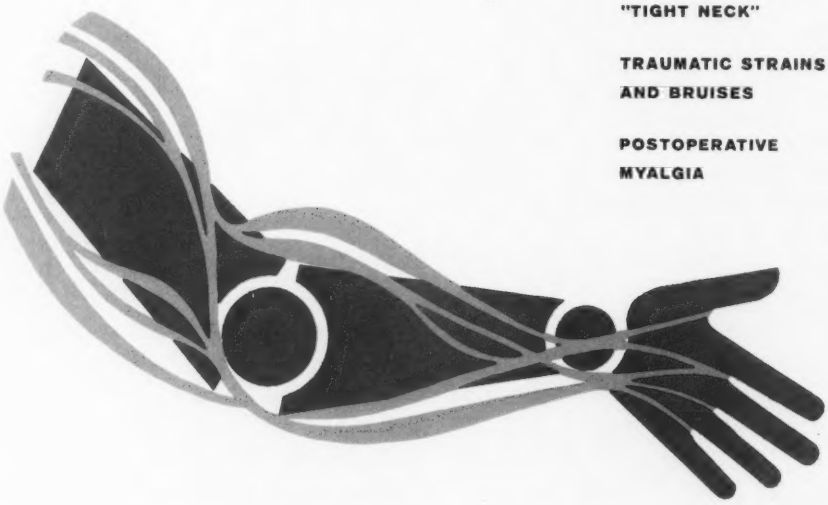
SPRAINED BACK

"TIGHT NECK"

TRAUMATIC STRAINS AND BRUISES

POSTOPERATIVE MYALGIA



■ Exhibits unusual analgesic properties, different from those of any other drug ■ Specific and superior in relief of SOMAtic pain ■ Modifies central perception of pain without abolishing natural defense reflexes ■ Relaxes abnormal tension of skeletal muscle

SOMA™

N-isopropyl-2-methyl-2-propyl-1, 3-propanediol dicarbamate

■ More specific than salicylates ■ Less drastic than steroids

■ More effective than muscle relaxants

SOMA has an unique analgesic action. It apparently modifies central pain perception without abolishing peripheral pain reflexes. SOMA is particularly effective in relieving joint pain. Patients say that they feel better and sleep better with SOMA than with previously used analgesic, sedative or relaxant drugs.
SOMA also relaxes muscle hypertonia, with its stresses on related joints, ligaments and skeletal structures.

ACTS FAST. Pain-relieving and relaxant effects start in 30 minutes and last 6 hours.

NOTABLY SAFE. Toxicity of SOMA is extremely low. No effects on liver, endocrine system, blood pressure, blood picture or urine have been reported. Some patients may become sleepy, particularly on high dosage.

EASY TO USE. Usual adult dose is one 350 mg. tablet 3 times daily and at bedtime.

SUPPLIED: Bottles of 50 white coated 350 mg. tablets.

Literature and samples on request.

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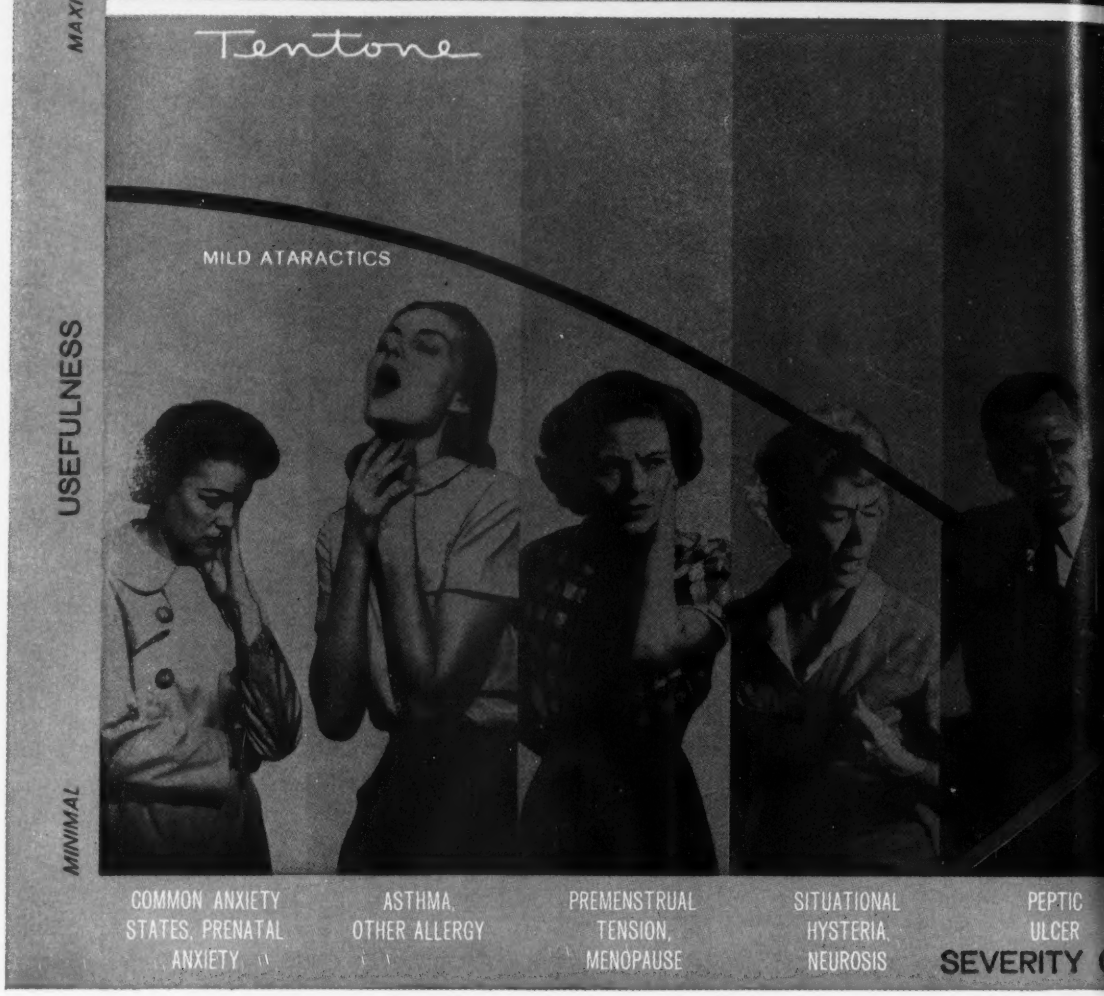
when a tranquilizer is warranted..



The extended usefulness of TENTONE is readily apparent

TENTONE® Methoxypromazine Maleate is a new, distinctive phenothiazine... highly active ...for general use in mild and moderate emotional and psychosomatic disorders.

TENTONE elicits a striking, positive calming response[1,2]...with marked reduction of psychic disorientation, and low risk of blood, liver or other organic toxicity and intolerance.[1-4]

TENTONE parallels the weaker ataractics in low incidence of side effects. Freedom from induced depression is apparently even greater.[5]

TENTONE provides a broadly adaptable dosage range (30 to 500 mg. daily) to permit maximum control in cases of varying severity.

TENTONE is also indicated to relieve emotional stress in surgical, obstetric and other hospitalized patients.

osage: Mild to moderate cases—average starting dose, one 10 mg. or one 25 mg. tablet ree or four times daily. *Moderate to severe*—average starting dose, one 50 mg. tablet ur times daily. *Supplied:* 10 mg., 25 mg., and 50 mg. tablets.

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Tentone

thoxypromazine Maleate

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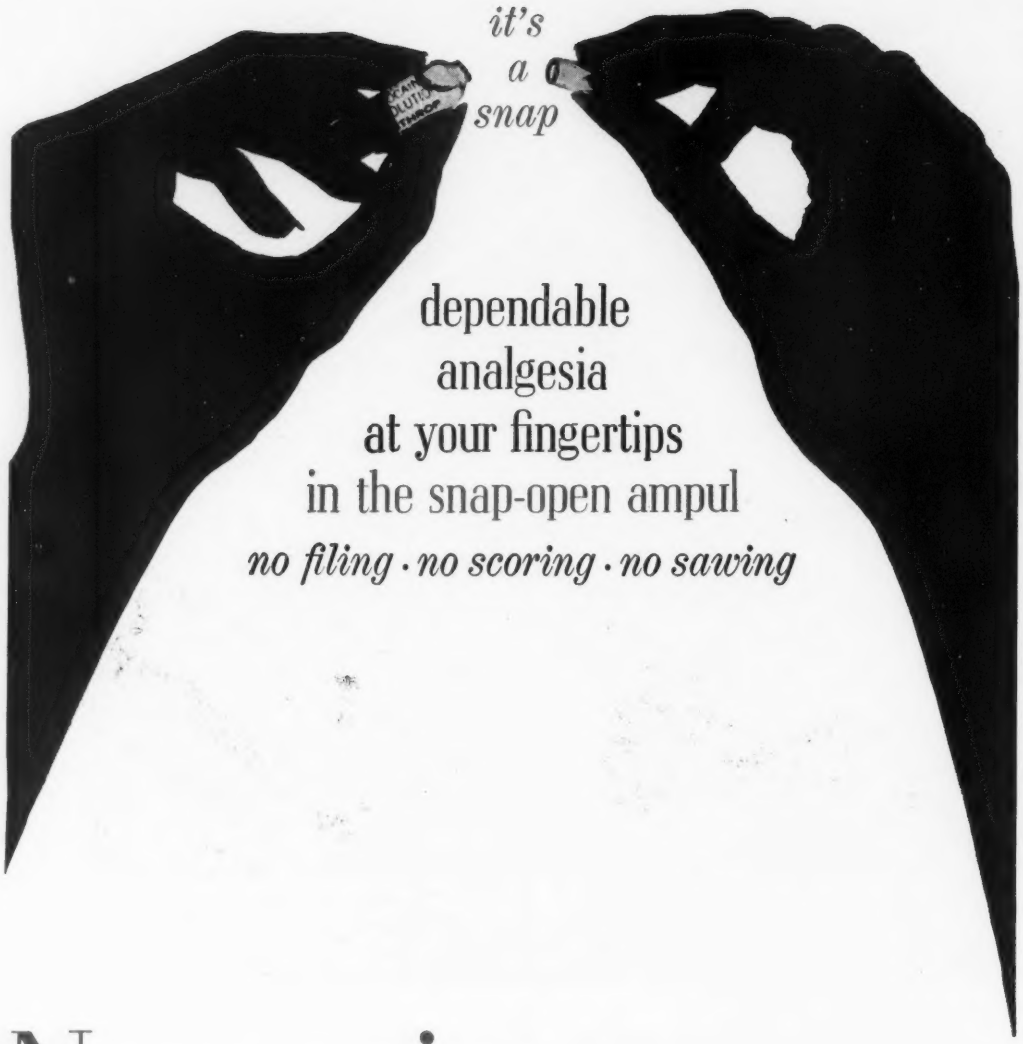

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analgesia
at your fingertips
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no filing · no scoring · no sawing

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the local anesthetic with universal acceptance

Novocain 1%, 2%, 10%, 20% Solutions with or without vasoconstrictors.
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Eyes Right!

the advantages of oil suspension
rapid even coverage on eye, lids, fornices . . . resists dilution by lacrimation . . . maintains effective antibiotic concentrations

the effectiveness of ACHROMYCIN
rapid suppression of common cocci and bacilli and of susceptible viruses—whether the primary infection or a complication of irritation, trauma, or inflammatory disease . . . fast resolution of swelling, erythema, and lesions . . . excellently tolerated

in the unique dropper-bottle
precise measurement of dose . . . clean . . . minimizes contamination . . . 4 cc. plastic squeeze dropper-bottle; 10 mg. (1%) ACHROMYCIN Tetracycline HCl per cc. sesame oil suspension

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OPHTHALMIC OIL SUSPENSION 1%

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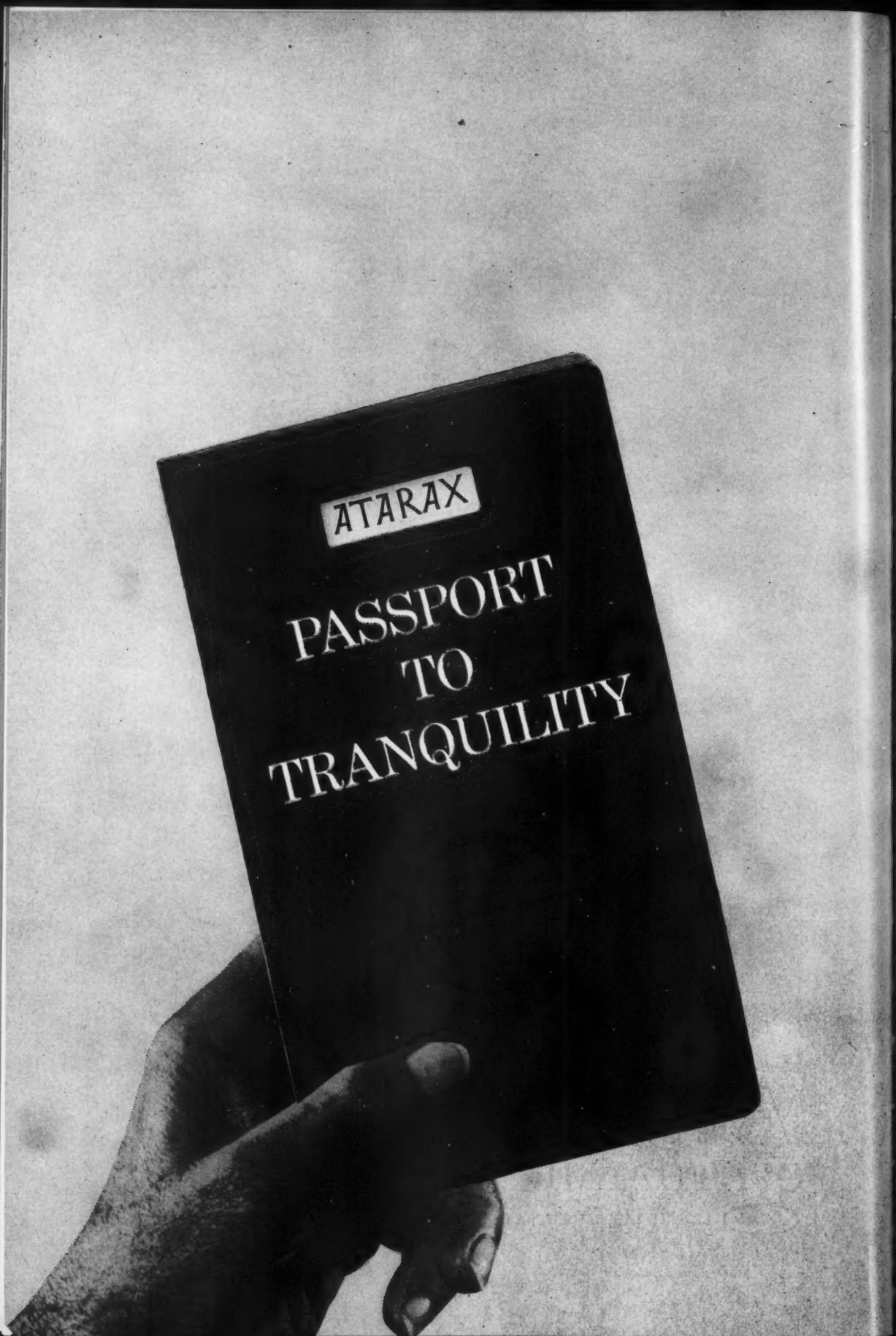
ATARAX
PASSPORT
TO
TRANQUILITY

Passport

☞ **a universal record of effectiveness**

In anxiety, tension and agitation, ATARAX "... produced a more favorable state of calm and tranquility than any drug previously used."[1]

☞ **widest latitude of safety and flexibility**

No serious adverse reaction ever documented – five dosage forms and sizes

☞ **chemically distinct among tranquilizers**

Not a phenothiazine or a meprobamate

☞ **added frontiers of usefulness**

These unique benefits in specific indications

ANTIHISTAMINIC
ANTIARRHYTHMIC
ANTISECRETORY

BACKED BY A WORLD OF EXPERIENCE

Dosage: ADULTS, one 25 mg. tablet, or one tbsp. Syrup q.i.d. CHILDREN – 3-6 years, one 10 mg. tablet or one tsp. Syrup t.i.d.; over 6 years, two 10 mg. tablets or two tsp. Syrup t.i.d.

Supplied: Tiny 10 mg., 25 mg., and 100 mg. tablets, bottles of 100. Syrup, pint bottles. Parenteral Solution, 10 cc. multiple-dose vials.

References: 1. Farah, L.: Internat. Rec. Med. *169*:379 (June) 1956. 2. Smigel, J. O., et al.: J. Am. Geriatrics Soc. *7*:61 (Jan.) 1959. 3. Feinberg, A. R., et al.: J. Allergy *29*:358 (July) 1958. 4. Eisenberg, B. C.: J.A.M.A. *169*:14 (Jan. 3) 1959. 5. Maryssael, L.: Bruxelles-méd. *38*:141 (Jan. 26) 1958. 6. Pfleger, R.: Med. Klin. *53*:1030 (June 5) 1958. 7. Over 200 laboratory and clinical papers from 14 countries.

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(brand of hydroxyzine)

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Division, Chas. Pfizer & Co., Inc.
Science for the World's Well-Being

all three
of the controls
you want for
G-I spasm
and pain

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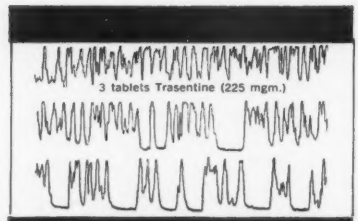
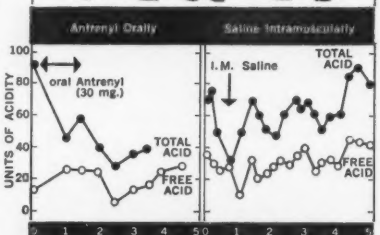
Trasentine-PA T.M.

Remember SERPASIL® (rescinnamine CIBA) for the anxious hypertensive with or without tachycardia

Reduction in degree of motility and tone, and return toward periodicity—in segment of descending human colon after Trasentine. (Adapted from Atkinson, et al.[1])



Average of effects of Antrenyl and saline on gastric secretion in three patients. (Adapted from Mattman and Strutner[2])



The patient with gastrointestinal symptoms benefits from general sedation coordinated with specific gastrointestinal relief.

1. ANTISPASMODIC CONTROL... Trasentine provides direct relaxing action on smooth muscle, induces an atropine-like effect, relieves pain with anesthetic effect on gastric mucosa.

2. ANTISECRETORY CONTROL... Antrenyl significantly reduces both total and free gastric acid levels.

3. SEDATIVE CONTROL... Phenobarbital decreases nervousness and irritability, provides much needed mental and physical rest.

DOSAGE: One tablet 3 or 4 times daily.

SUPPLIED: TRASENTINE-PA *Tablets*, each containing 75 mg. Trasentine hydrochloride, 7½ mg. Antrenyl bromide and 20 mg. phenobarbital; bottles of 100.

REFERENCES: 1. Atkinson, A. J., Adler, H. F., and Ivy, A. C.: J.A.M.A. *121*:646 (Feb. 27) 1943.
2. Mattman, P. E., and Strutner, L.: Am. J. Digest. Dis. *20*:126 (May) 1953.

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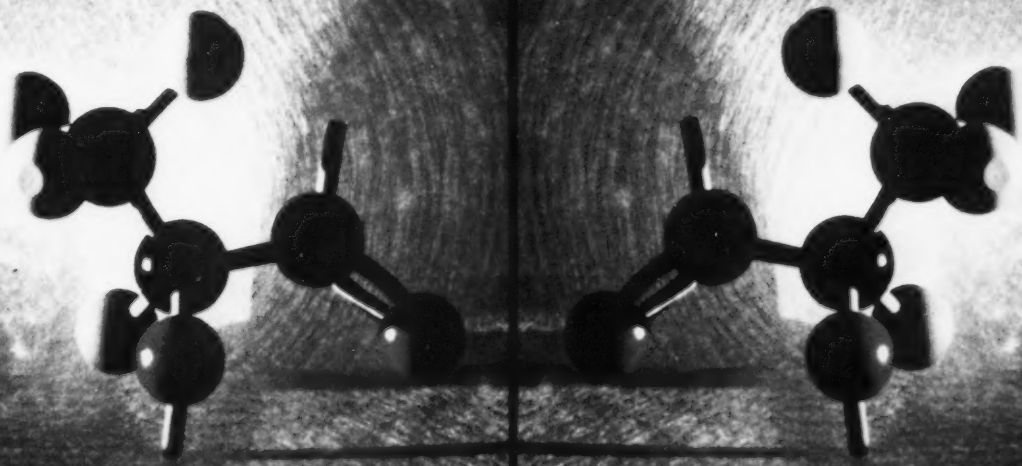
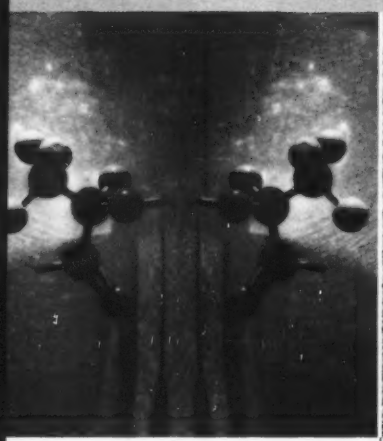
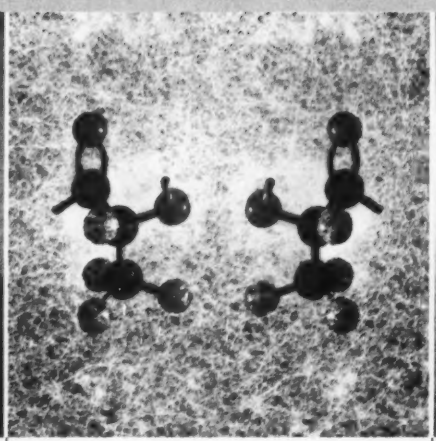


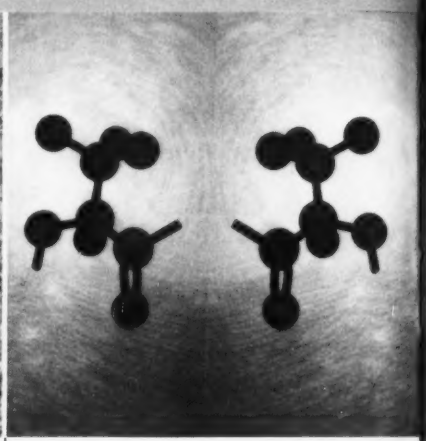
The first synthetic penicillin available for general clinical use

MAJOR THERAPEUTIC

SYNC







BLOOD LEVELS TWICE AS HIGH AS WITH POTASSIUM PENICILLIN V

SAFER ORAL ROUTE PROVIDES HIGHER BLOOD LEVELS THAN INTRAMUSCULAR PENICILLIN G

IMPROVED ANTIBIOTIC EFFECT FROM COMPLEMENTARY ACTION OF ISOMERS

ADVANTAGES ACCOMPANY MOLECULAR ASYMMETRY

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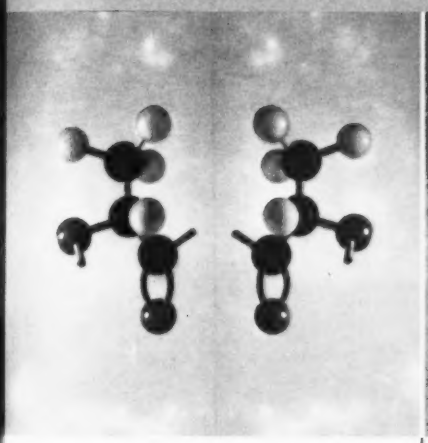
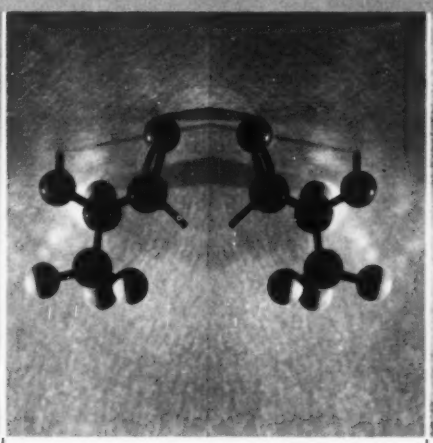
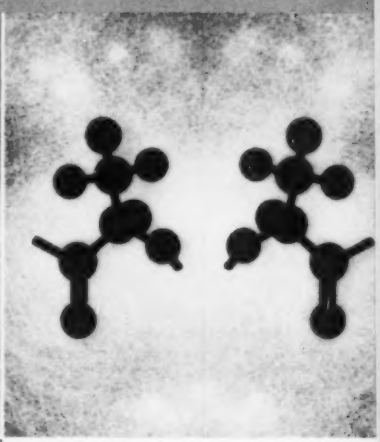
POTASSIUM PENICILLIN-152







ANTIBIOTIC ACTIVITY DIRECTLY PROPORTIONAL TO ORAL DOSE

REDUCED HAZARD OF SERIOUS ALLERGENICITY BY SAFER ORAL ROUTE

MANY STAPH STRAINS MORE SENSITIVE TO SYNCILLIN

BRISTOL

ORIGIN OF A NEW SYNTHETIC PENICILLIN

In March, 1957, Dr. John C. Sheehan of the Massachusetts Institute of Technology announced the total synthesis of penicillin from common raw materials, thus solving a problem which had baffled research workers for more than 15 years. Although total synthesis was not commercially practicable, this work, sponsored by Bristol Laboratories, made possible the subsequent synthesis of new penicillins not occurring in nature. Later scientists at Beecham Laboratories in England discovered that a key intermediate (6-aminopenicillanic acid) could be produced by a fermentation process. With these achievements, large scale production of synthetic penicillins became feasible.

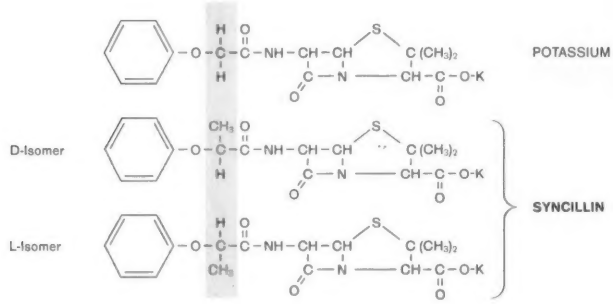
Organic chemists at Bristol then embarked upon an intensive program to develop better penicillins. Over five hundred were synthesized and underwent preliminary screening. Forty-six showed sufficient promise to warrant further investigation. Extensive microbiological, pharmacological, and clinical screening indicated that one compound, SYNCILLIN, had advantages of major importance over other penicillins.

SYNCILLIN is the N-acylation product of 6-aminopenicillanic acid and α-phenoxypropionic acid (the phenylether of lactic acid). It is freely soluble in water and remarkably resistant to decomposition by acid. The acid stability of SYNCILLIN is equivalent to that of penicillin V at pH 2 and pH 3 at 37° C.[1]

SIGNIFICANCE OF MOLECULAR ASYMMETRY AND ISOMERIC COMPLEMENTARITY

SYNCILLIN has a molecular configuration similar to penicillin V, but contains an additional CH_3 group so positioned as to render the adjacent carbon atom asymmetric. (In the formulae below, the added CH_3 group is shown in blue and the asymmetric carbon atom in red.) As a result, SYNCILLIN occurs as a mixture of two isomers.

Each isomer has been synthesized in essentially pure form and found to possess distinctive chemical and biological properties. The L-isomer is 2 to 17 times more active than the D-isomer against many of the organisms tested. As produced, SYNCILLIN is a mixture of the L-isomer and the D-isomer. As will be shown later, the antibiotic effect of the clinically available mixture, SYNCILLIN, is greater than either isomer alone against many organisms. This phenomenon is referred to here as *isomeric complementarity*.



SYNCILLIN

major therapeutic advantages accompany molecular asymmetry

ISOMERIC COMPLEMENTARITY DEMONSTRATED IN VITRO

The *in vitro* minimum inhibitory concentration (MIC) of SYNCILLIN and of each of its two component isomers was determined for a variety of common pathogens and laboratory test organisms. As may be seen from Table 1, all three are highly effective against penicillin-susceptible staphylococci and against pneumococci, streptococci, gonococci, and corynebacteria; all are ineffective against Salmonella, *E. coli,* and other gram-negative coliform bacilli.

SYNCILLIN was more active against many of the test strains including some streptococci and staphylococci than either of its components. This demonstrates *in vitro* the phenomenon of isomeric complementarity.

TABLE 1

Minimum Concentrations of SYNCILLIN and Components Required to Inhibit a Wide Range of Bacteria

SYNCILLIN

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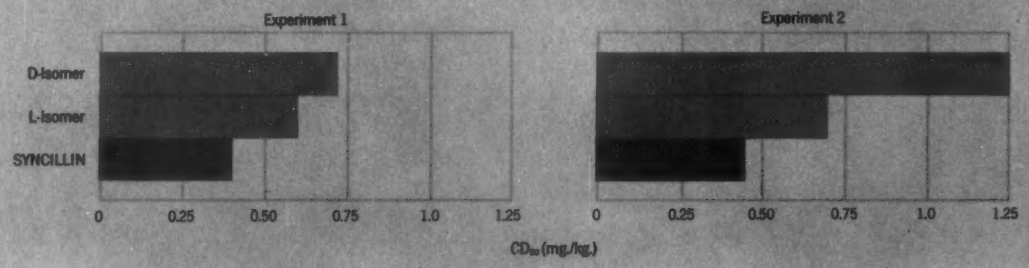
ISOMERIC COMPLEMENTARITY CONFIRMED IN VIVO

To determine the median curative dose (CD_{50}) mice were infected with 100 times the lethal dose of *Staphylococcus aureus*. Each penicillin being tested was administered intramuscularly at the same time, and the dose required to cure half the animals determined. The greater effect of the mixture of the two isomers (SYNCILLIN) is shown in two independent experiments. (See Figure 1.) Note that isomeric complementarity is thus confirmed *in vivo*.

FIGURE 1 — Median Curative Dose (CD_{50}) for *Staphylococcus aureus* (var. Smith) Infections

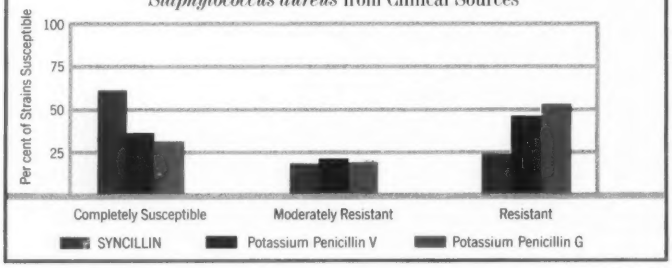


MANY STRAINS OF STAPHYLOCOCCI MORE SENSITIVE TO SYNCILLIN

SYNCILLIN has been tested against a large number of strains of *Staphylococcus aureus* isolated from clinical sources. Many organisms resistant to potassium penicillin G and potassium penicillin V proved sensitive to SYNCILLIN.

Wright[2] performed sensitivity studies on 54 strains, the majority of which were resistant or moderately resistant to penicillin V and penicillin G. Thirty-two (60%) of the strains were sensitive to SYNCILLIN, approximately twice as many as with the other penicillins. (See Figure 2.) In two-thirds of the isolates, SYNCILLIN produced inhibition at concentrations lower than those required for either of the other antibiotics. One strain was more sensitive to penicillin G.

FIGURE 2 – *In Vitro* Sensitivity of 54 Strains of Coagulase-Positive *Staphylococcus aureus* from Clinical Sources

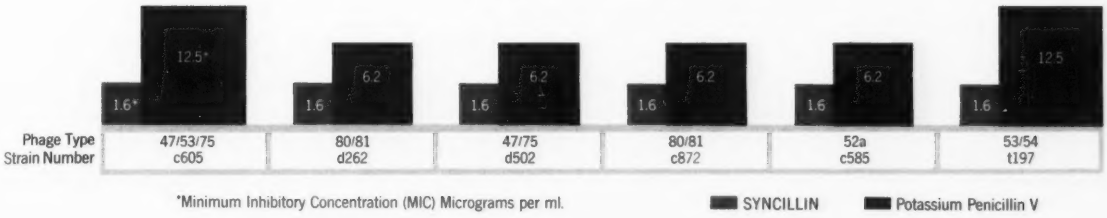


Adapted from Wright[2]

Of equal interest are the findings of White.[3] Six penicillin-resistant strains of staphylococci were isolated from hospital infections. None was sensitive to potassium penicillin V. All were sensitive to SYNCILLIN. (See Figure 3.)

FIGURE 3

Minimum Concentrations of SYNCILLIN Required to Inhibit
Hospital Strains of *Staphylococcus aureus* Resistant to Potassium Penicillin V

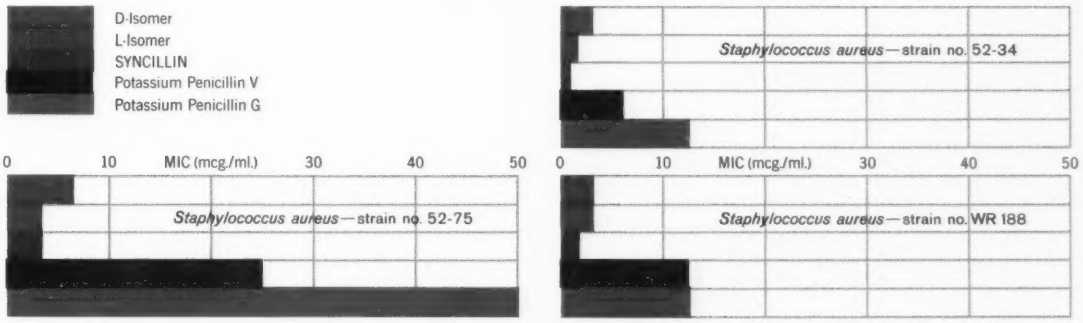


The efficacy of SYNCILLIN against the type 80/81 Staphylococcus (dangerous and widespread in hospitals) is worthy of special attention.

The complementary action of the component isomers is also seen with strains of staphylococci resistant to penicillins. Note that SYNCILLIN is more effective than either isomer against strains 52-34 and WR 188. (See Figure 4.) Against all three strains, SYNCILLIN is effective at concentrations below serum levels, while penicillins V and G are ineffective.

FIGURE 4

Minimum Inhibitory Concentrations (MIC) for Coagulase-Positive
Penicillin-Resistant Strains of *Staphylococcus aureus*



Isomeric complementarity has thus been demonstrated for:

— *certain penicillin-susceptible streptococci, staphylococci and corynebacteria* in vitro *(Table 1)*

— *penicillin-susceptible staphylococci* in vivo *(Figure 1)*

— *penicillin-resistant staphylococci* in vitro *(Figure 4)*

SYNCILLIN

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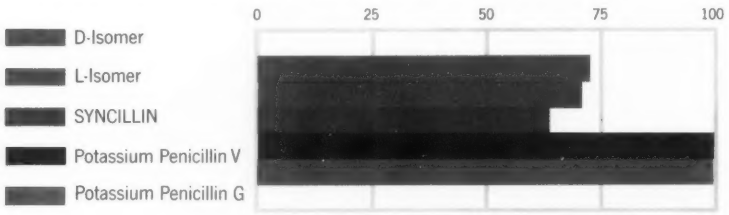
ISOMERIC COMPLEMENTARITY SHOWN BY REDUCED RATE OF INACTIVATION BY PENICILLINASE

Bacterial resistance to penicillin has been attributed to the action of penicillin-inactivating enzymes produced by the invading organisms.[4] As shown in Figure 5, SYNCILLIN is less affected by staphylococcal penicillinase than either of its component isomers – a further demonstration of isomeric complementarity. Further, SYNCILLIN is shown to be less inactivated by this enzyme than penicillin V and penicillin G.

Resistance to SYNCILLIN develops in a slow, step-wise manner characteristic of other penicillins, in contrast to the usually rapid development of resistance to streptomycin.

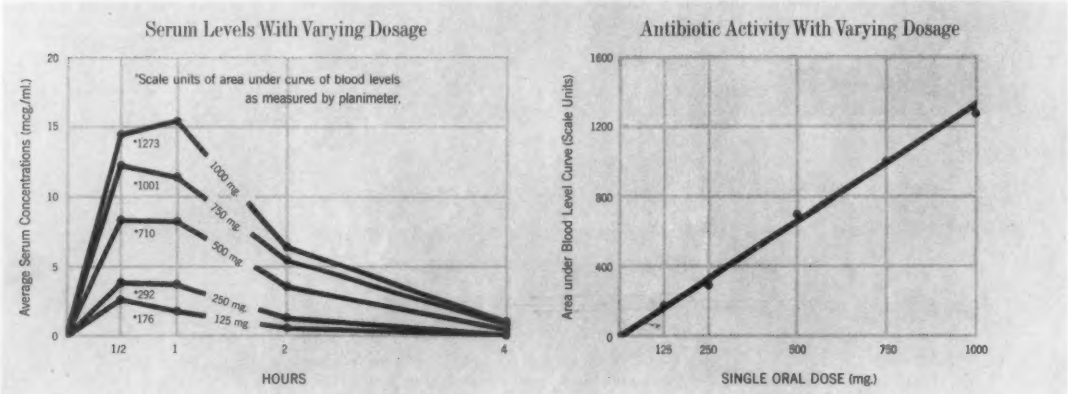
FIGURE 5—Effect of Staphylococcal Penicillinase on Different Penicillins



ANTIBIOTIC ACTIVITY DIRECTLY PROPORTIONAL TO ORAL DOSAGE

Cronk[5] studied blood levels after administering varying amounts of SYNCILLIN. (Figure 6.) Total antibiotic activity (obtained by measuring areas under curves with a planimeter) increases rapidly as the dose is doubled. These data show that increased dosage markedly increases serum concentration and thus may enhance the drug's effectiveness.

FIGURE 6



SYNCILLIN

major therapeutic advantages accompany molecular asymmetry

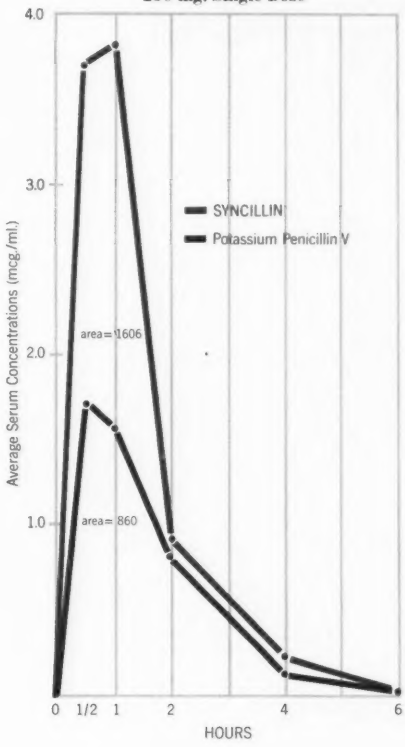
BLOOD LEVELS TWICE AS HIGH AS WITH POTASSIUM PENICILLIN V AFTER ORAL ADMINISTRATION

Wright[6] performed comparative crossover blood level studies on volunteer subjects receiving equivalent amounts of potassium penicillin V and SYNCILLIN. The peak concentrations attained during the first hour after administration were twice as high with SYNCILLIN.

The total antibiotic activity as measured by the area under the curves (see Figure 7) indicates an almost 2 to 1 superiority of SYNCILLIN (1606) over potassium penicillin V (860).

The higher blood levels may be of value with organisms of only moderate penicillin-sensitivity where doubling the blood concentration may be essential for effective bactericidal action. In addition these higher levels may be necessary where there is infection in areas with a poor blood supply.[7] Under these circumstances a higher blood concentration may provide the increased diffusion pressure required to deliver adequate amounts to the tissue.

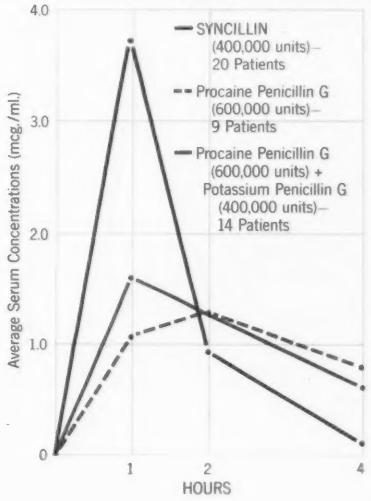
FIGURE 7
20 Subject Crossover
250 mg. Single Dose



BLOOD LEVELS MUCH HIGHER THAN WITH INTRAMUSCULAR PENICILLIN G

In addition, blood levels attained with oral SYNCILLIN[6] are much higher than those with intramuscular penicillin G.[8a, b] (See Figure 8.) Note that the level at one hour for SYNCILLIN (3.8 mcg./ml.) is more than twice as high as with procaine penicillin G, even when reinforced with potassium penicillin G (1.6 mcg./ml.). Since penicillins are *bactericidal,* these intermittent high serum levels can be clinically significant. Thus, SYNCILLIN offers the promise of superior efficacy via the safer oral route.

FIGURE 8—Serum Levels after Oral Administration of SYNCILLIN (250 mg.) and after Intramuscular Injection of Penicillin G



SYNCILLIN

major therapeutic advantages accompany molecular asymmetry

REDUCED HAZARD OF SERIOUS ALLERGENICITY BY SAFER ORAL ROUTE

SYNCILLIN has been administered in multiple doses to 437 patients and volunteers. One patient developed itching during therapy, possibly an allergic side effect. Another had a purpuric rash, but no relationship to SYNCILLIN was established. No reactions were observed in 9 patients with a known history of sensitivity to penicillin.

While the above data suggests the possibility of reduced allergenic hazard, no definite conclusions may be drawn at this time. *The usual precautions for oral penicillin therapy should be observed.* Patients with histories of asthma, hay fever, urticaria, or previous penicillin-sensitivity should especially be watched carefully. Since SYNCILLIN is administered orally, it may be expected to be safer than parenteral penicillin.

As Flippin[9] recently stated, "... it is well established that serious allergy to the drug [penicillin] is most likely to occur following parenteral administration, especially after repeated intramuscular injections; the oral route is least likely to initiate severe hypersensitivity reactions. This can be explained partly by the fact that when reactions develop following oral medication, they are usually slow enough to treat symptomatically; thus the progression of the reaction can usually be interrupted.... In view of the relatively high incidence of severe allergy to injectable penicillin, it would seem advisable to employ oral penicillin routinely, except in the control of infections involving the blood stream, endocardium, meninges, etc., in which cases the parenteral route remains the preferred treatment."

SYNCILLIN, like other penicillins, is essentially free of other toxicity. No hematopoietic, hepatic, or renal toxicity was observed in 210 volunteers receiving 1 gm. daily for 2 to 3 weeks.[10]

CLINICAL EFFICACY DEMONSTRATED IN PENICILLIN-SENSITIVE INFECTIONS

Clinical trials conducted by Blau and Kanof,[11] White,[12] Prigot,[13] Robinson,[14] Dube,[15] Ferguson,[16] Rutenburg,[17] Richardson,[18] Bunn,[19] Cronk,[5] Kligman,[10] and Yow [20] demonstrated the efficacy of SYNCILLIN in a variety of streptococcal, staphylococcal, pneumococcal, and gonococcal infections. Conditions treated included respiratory, skin, soft tissue, wound, and chronic urinary tract infections; acute gonorrhea; cellulitis; septicemia; otitis media; gingivitis; and Vincent's angina. In a few patients SYNCILLIN was used for rheumatic fever or gonorrheal prophylaxis.

One hundred seventy-two of one hundred ninety-six patients responded favorably to SYNCILLIN. The failures included 1 patient with pustular dermatoses, 10 elderly patients with chronic urinary tract infections, 1 patient with gonorrhea, 1 patient with a gram-negative infection, and 10 patients with staphylococcal infections. Lack of response of staphylococcal infections was attributed to the presence of resistant organisms or local suppurative foci requiring drainage.

SYNCILLIN

major therapeutic advantages accompany molecular asymmetry

Relatively few side effects were encountered. One patient experienced moderate itching of the skin which was controlled by an antihistamine. Another reported pruritus ani which did not interfere with therapy. Diarrhea occurred in 4 instances. There was one purpuric rash, but no relationship to SYNCILLIN could be established.

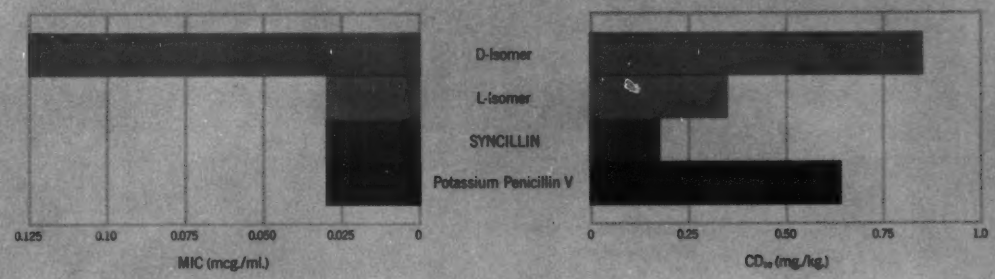
Clinical response usually begins within 24 hours in infections susceptible to SYNCILLIN. Recovery occurs in 4 to 7 days depending upon the severity of the infection. Gonorrheal infections respond very promptly to SYNCILLIN; 500 mg. b.i.d. for two days usually produce bacteriologic cures.

IMPROVED ANTIBIOTIC EFFECT FROM COMPLEMENTARY ACTION OF ISOMERS

SYNCILLIN is a mixture of isomers. The L-isomer is 2 to 17 times more active than the D-isomer against many of the organisms tested. Furthermore, the D- and L-isomers have other distinguishing chemical, pharmacological, and microbiological properties. Their *in vivo* and *in vitro* activities differ for many important pathogens. *Against many of the organisms tested, the combination of isomers* (SYNCILLIN) *is much more active than the stronger isomer alone.* This phenomenon of isomeric complementarity is not always demonstrable, for in a few instances SYNCILLIN is slightly less active.

Isomeric complementarity has previously been demonstrated *in vitro* (Figure 4) and *in vivo* (Figure 1). Figure 9 reveals a third form of superiority related to isomeric complementarity. Equal concentrations of SYNCILLIN and penicillin V were required to inhibit this growth of staphylococci *in vitro.* But, *in vivo,* a much smaller amount of SYNCILLIN *(one-third that of penicillin V)* was effective in an experimental infection with the same strain. These observations on complementary action indicated the advantage of producing the mixture of isomers as the medication to be made available for clinical therapy.

FIGURE 9 — Comparison of CD_m and MIC Values Against *Staphylococcus aureus* (var. Smith)



Isomeric complementarity has thus been demonstrated for:

— *certain penicillin-susceptible streptococci, staphylococci and corynebacteria* in vitro *(Table 1)*

— *penicillin-susceptible staphylococci* in vivo *(Figures 1 and 9)*

— *penicillin-resistant staphylococci* in vitro *(Figure 4)*

— *staphylococcal penicillinase antibiotic inactivation (Figure 5)*

SYNCILLIN

major therapeutic advantages accompany molecular asymmetry

Indications:

SYNCILLIN is recommended in the treatment of infections caused by pneumococci, streptococci, gonococci, corynebacteria, and penicillin-sensitive staphylococci. In addition, SYNCILLIN is effective against certain strains of staphylococci resistant to other penicillins.

SYNCILLIN, like other oral penicillins, is not recommended at the present time in deep-seated or chronic infections, subacute bacterial endocarditis, meningitis, or syphilis.

Dosage:

125 mg. or 250 mg. three times daily, depending on the severity of infection. Larger doses (e.g., 500 mg. t.i.d.) may be used for more severe infections. SYNCILLIN may be administered without regard to meals.

Beta hemolytic streptococcal infections should be treated with SYNCILLIN for at least ten days.

Precautions:

While present data suggest the possibility of reduced allergenic hazard, no definite conclusions may be drawn at this time. *Therefore the usual precautions with oral penicillin therapy must be observed.* Patients with histories of asthma, hay fever, urticaria, or previous reactions to penicillin should be watched with special care.

Diarrhea has been reported occasionally following heavy dosage. If this occurs, the interval between dosages should be lengthened.

If superinfection occurs during therapy, appropriate measures should be taken.

Since some strains of staphylococci are resistant to SYNCILLIN as well as to other penicillins, cultures and sensitivity tests should be performed where indicated by clinical judgment. As is true with all antibiotics, clinical response does not always correlate with laboratory bacterial sensitivity reports.

Supply:

125 and 250 mg. tablets, bottles of 25 and 100. 125 mg. powder for oral solution, 60 ml. vials.

References: 1. Lein, J.: Microbiology report to Bristol Laboratories Inc. 2. Wright, W. W.: Microbiology report to Bristol Laboratories Inc. 3. White, A. C.: Microbiology report to Bristol Laboratories Inc. 4. Dubos, R. J.: Bacterial and Mycotic Infections of Man, 3rd edition, Philadelphia, J. B. Lippincott Co., p. 690. 5. Cronk, G. A.: Clinical report to Bristol Laboratories Inc. 6. Wright, W. W.: Clinical report to Bristol Laboratories Inc. 7. Kass, E. H.: Am. J. Med. *18*:764 (May) 1955. 8a. White, A. C.; Couch, R. A.; Foster, F.; Calloway, J.; Hunter, W., and Knight, V.: in Welch, H. and Marti-Ibañez, F.: Antibiotics Annual — 1955-1956, Medical Encyclopedia, Inc., New York, 1956, p. 490. b. Data on file — at Bristol Laboratories. 9. Flippin, H. F.: Pennsylvania M. J. *62*:864 (June) 1959. 10. Kligman, A.: Clinical report to Bristol Laboratories Inc. 11. Blau, S., and Kanof, N.: Clinical report to Bristol Laboratories Inc. 12. White, A. C.: Clinical report to Bristol Laboratories Inc. 13. Prigot, A.: Clinical report to Bristol Laboratories Inc. 14. Robinson, C.: Clinical report to Bristol Laboratories Inc. 15. Dube, A. H.: Clinical report to Bristol Laboratories Inc. 16. Ferguson, B.: Clinical report to Bristol Laboratories Inc. 17. Rutenburg, A. M.: Clinical report to Bristol Laboratories Inc. 18. Richardson, J. H.: Clinical report to Bristol Laboratories Inc. 19. Bunn, P. A.: Clinical report to Bristol Laboratories Inc. 20. Yow, E. M.: Clinical report to Bristol Laboratories Inc.

BRISTOL

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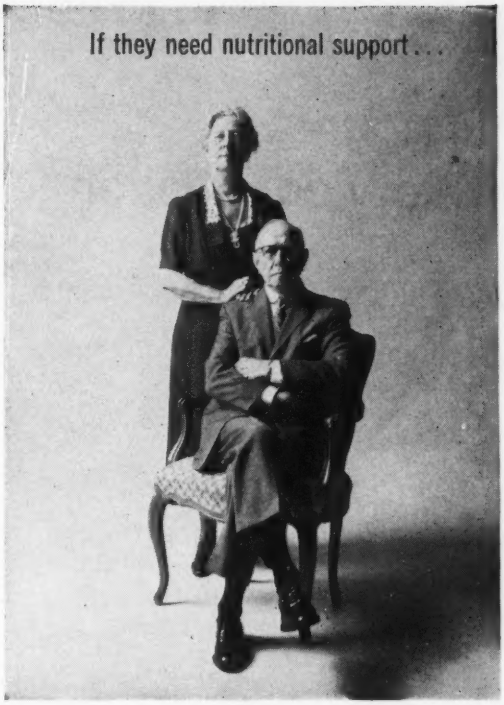
SYNCILLIN



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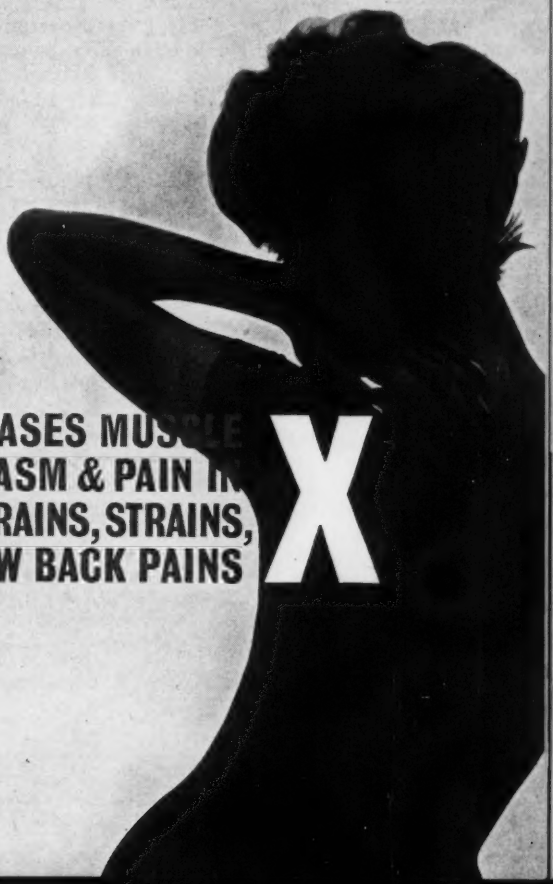

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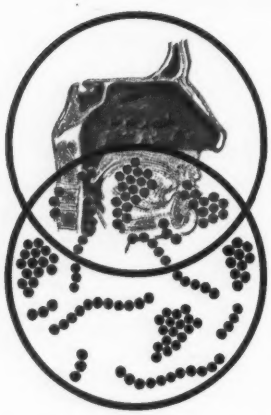

when upper respiratory congestion is complicated by bacterial invaders

TRISULFAMINIC provides logical therapy

- for the patient ill with congestion and infection of the upper respiratory tract, as in purulent rhinitis, sinusitis, tonsillitis and otitis media, when caused by sulfa-susceptible bacteria;
- because secondary invasion by such bacteria so frequently follows the common cold.[1]

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Triaminic and triple sulfas are not only pharmacologically *compatible*, they are a therapeutically *logical* combination for upper respiratory infections: Triaminic for effective decongestant relief from rhinitis, rhinorrhea and sinusitis;[2] triple sulfas for well-established antibacterial action.

The advantages of Trisulfaminic in upper respiratory infections include: proved effectiveness; safety; economy; ease of administration; less likelihood of sensitivity reactions;[3] compatibility with antibiotics and other antibacterial therapy. Provided also as Suspension for additional convenience.

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Available as TABLETS and SUSPENSION

Each easy-to-swallow Trisulfaminic Tablet or 5 ml. teaspoonful of Suspension provides:

Triaminic® .....25 mg.
(phenylpropanolamine HCl 12.5 mg.
pheniramine maleate ..... 6.25 mg.
pyrilamine maleate ..... 6.25 mg.)
Trisulfapyrimidines, U.S.P. .....0.5 Gm.

Dosage:

Adults—2 to 4 tablets or tsp. initially, followed by 2 tablets or tsp. every 4 to 6 hours until the patient has been afebrile 3 days. *Children 8 to 12* — 2 tablets or tsp. initially, followed by 1 tablet or tsp. every 6 hours. *Children under 8*—dosage according to weight.

The palatability, convenience and effectiveness of the Suspension make it especially suitable for children and for those older patients who prefer liquid medication.

References: 1. Cecil, R. L., et al.: J.A.M.A. 124:8 (Jan. 1) 1944. 2. Fabricant, N. D.: E.E.N.T. Monthly 37:460 (July) 1958. 3. Beckman, H.: Drugs, Their Nature, Action & Use, Saunders, Philadelphia, 1958, p. 527.

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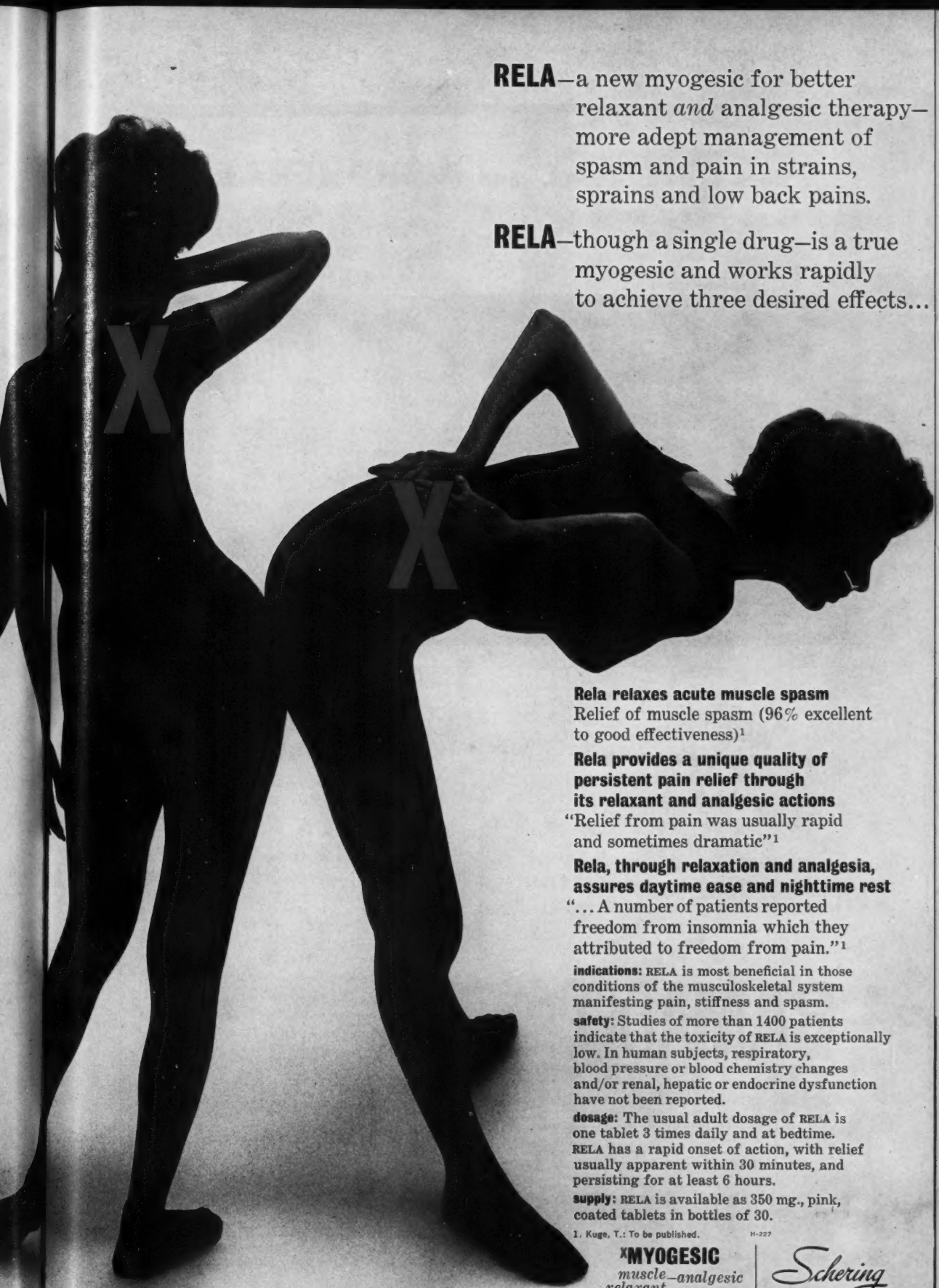

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supply: RELA is available as 350 mg., pink, coated tablets in bottles of 30.

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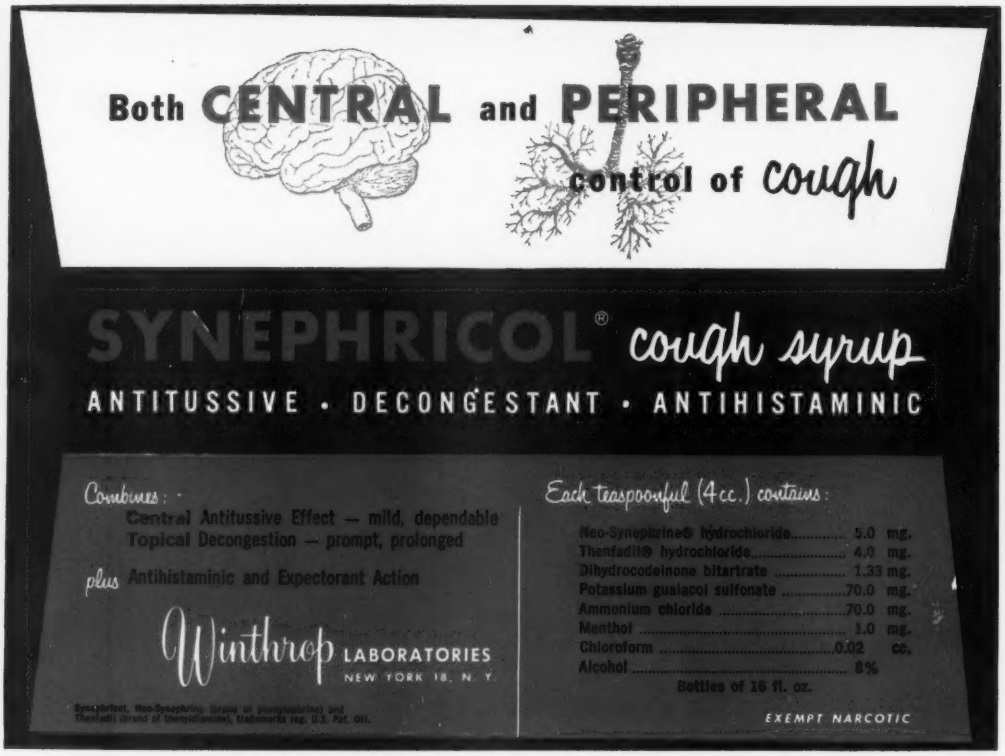
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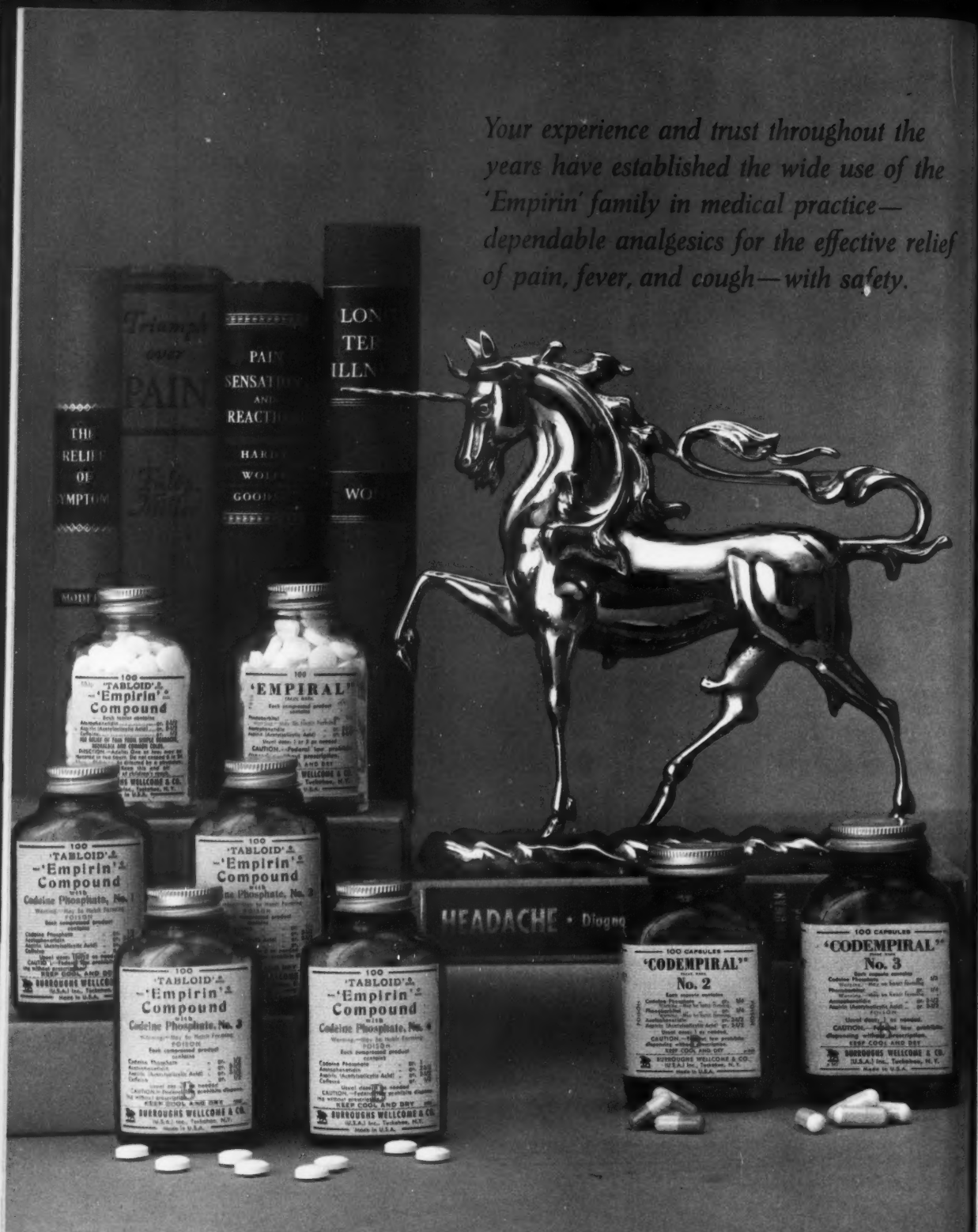
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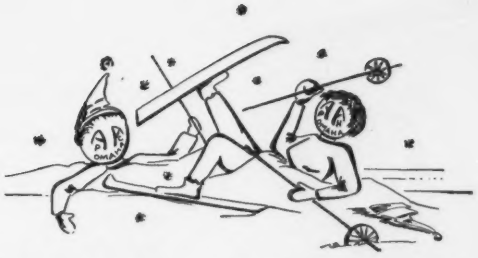

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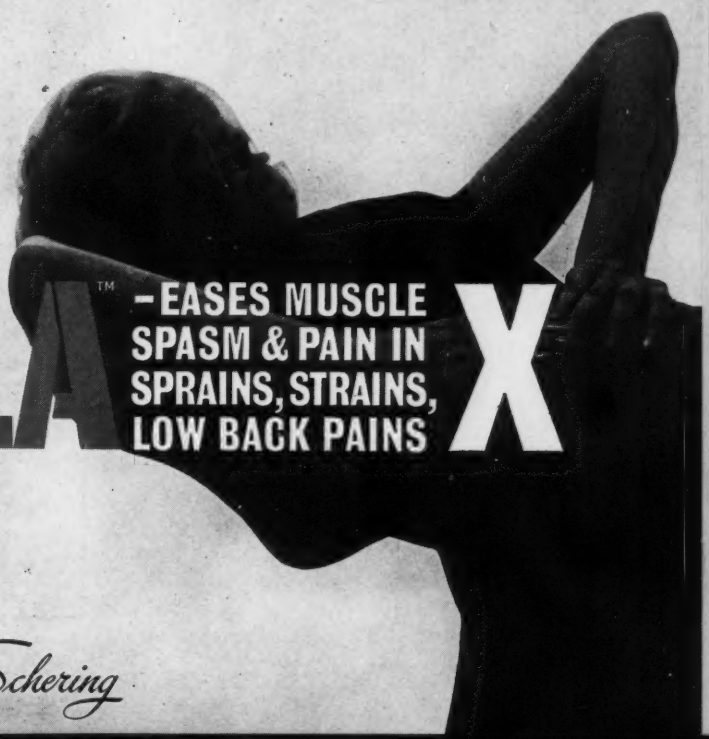

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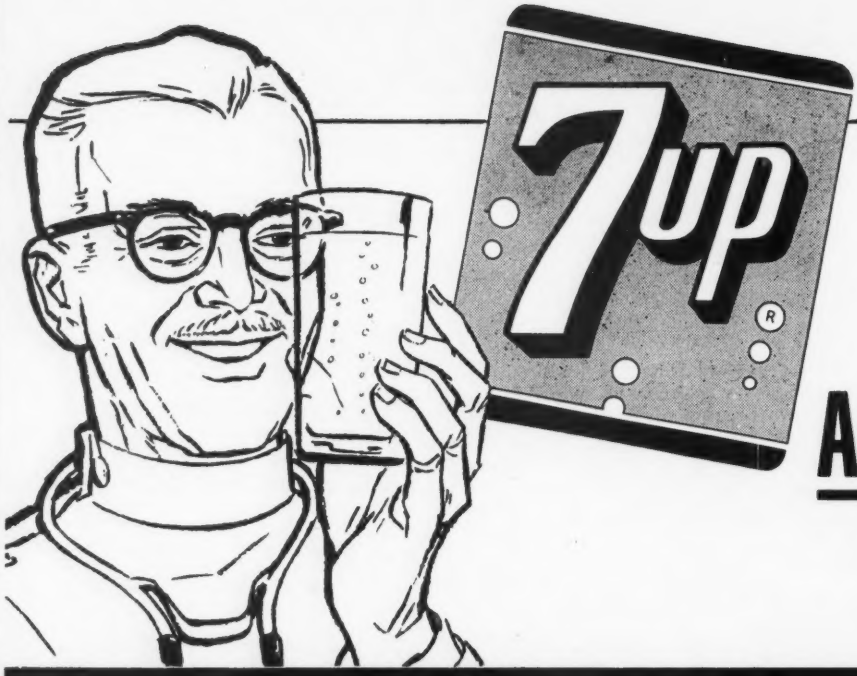

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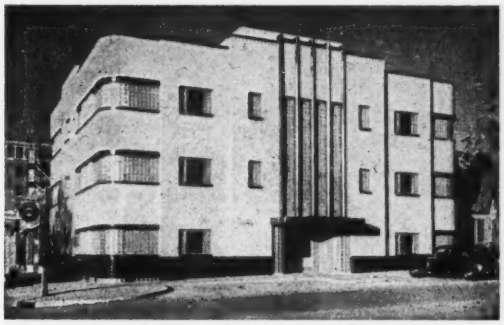

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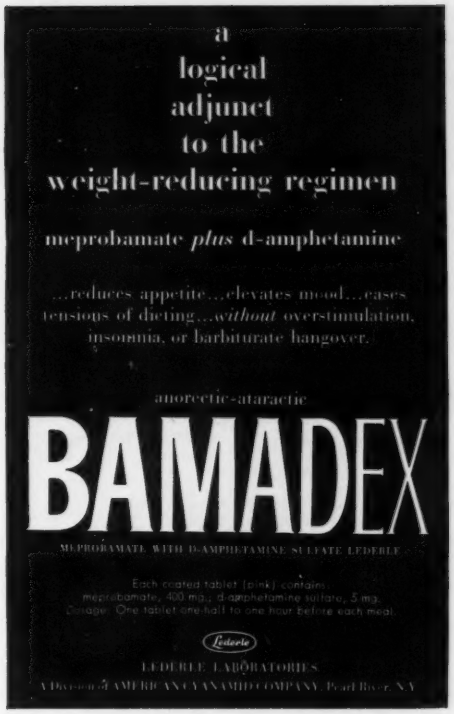
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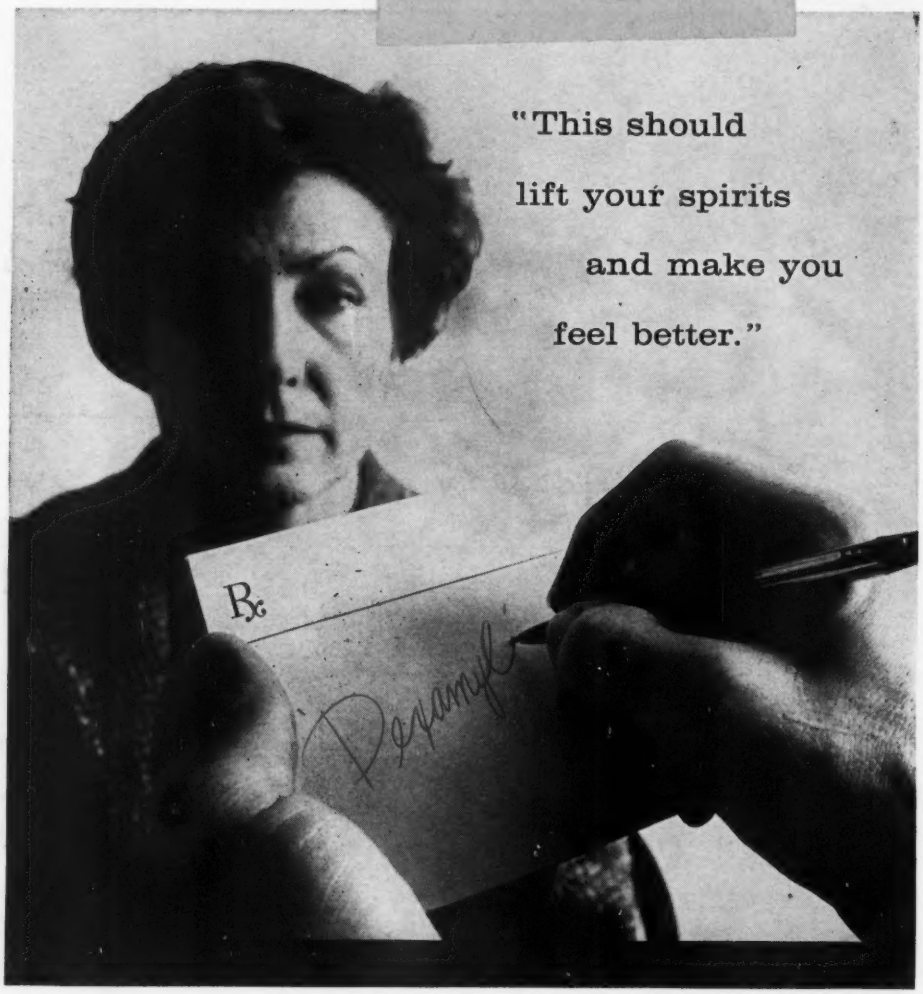

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